



CAUSATION, 17(12): 5–131

[DOI:10.5281/zenodo.6791972](https://doi.org/10.5281/zenodo.6791972)

Received: July 3, 2022

Accepted: July 3, 2022

Published: July 3, 2022

[Deutsche Nationalbibliothek Frankfurt](#)

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# Without Epstein Barr virus infection, no non Hodgkin lymphoma

*Research article*

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## Abstract

### Background:

Lymphomas are tumours of the immune system. About 10% of all lymphomas are Hodgkin's lymphoma (HL), while the remaining 90% are referred to as non-Hodgkin lymphoma (NHL). The cause or even a cause of NHL is still not identified.

### Methods:

View studies which investigated the relationship between Epstein-Barr virus (EBV) and NHL have been re-analysed.

### Results:

**Without** Epstein-Barr virus infection, **no** non-Hodgkin lymphoma (P Value = 0,0191030137).

### Conclusion:

Within Epstein-Barr virus, the cause of non-Hodgkin lymphoma will be found.

**Keywords:** Epstein-Barr virus; non-Hodgkin lymphoma; Necessary condition; Cause; Effect; Causal relationship k; Causality; Causation

## 1. Introduction

Non-Hodgkin's lymphoma (NHL)<sup>1</sup>, a neoplasm of the lymphoid tissues, is originating from mature B cells, B cell precursors, T cell precursors, and mature T cells. NHL is equally the most common<sup>2</sup> and very heterogeneous<sup>3, 4, 5</sup> hematological malignancy. Based on the disease's prognosis, Non-Hodgkin lymphoma is divided into two groups, "indolent" and "aggressive". NHL is determined

<sup>1</sup>Thandra KC, Barsouk A, Saginala K, Padala SA, Barsouk A, Rawla P. Epidemiology of Non-Hodgkin's Lymphoma. *Med Sci (Basel)*. 2021 Jan 30;9(1):5. doi: 10.3390/medsci9010005. PMID: 33573146; PMCID: PMC7930980.

<sup>2</sup>Jemal A, Bray F, Center MM, Ferlay J, Ward E, Forman D. Global cancer statistics. *CA Cancer J Clin*. 2011 Mar-Apr;61(2):69-90. doi: 10.3322/caac.20107. Epub 2011 Feb 4. Erratum in: *CA Cancer J Clin*. 2011 Mar-Apr;61(2):134. PMID: 21296855. Format:

<sup>3</sup>Jaffe ES, Harris NL, Stein H, Vardiman JW, eds. *World Health Organization Classification of Tumours of Haematopoietic and Lymphoid Tissues*. Lyon, France: IARC Press; 2001

<sup>4</sup>Swerdlow SH, Campo E, Harris NL, et al. eds. *World Health Organization Classification of Tumours of Haematopoietic and Lymphoid Tissues*. 4th ed. Lyon, France: IARC Press; 2008

<sup>5</sup>Morton LM, Sampson JN, Cerhan JR, Turner JJ, Vajdic CM, Wang SS, Smedby KE, de Sanjosé S, Monnereau A, Benavente Y, Bracci PM, Chiu BC, Skibola CF, Zhang Y, Mbulaiteye SM, Spriggs M, Robinson D, Norman AD, Kane EV, Spinelli JJ, Kelly JL, La Vecchia C, Dal Maso L, Maynadié M, Kadin ME, Cocco P, Costantini AS, Clarke CA, Roman E, Miligi L, Colt JS, Berndt SI, Mannetje A, de Roos AJ, Krickler A, Nieters A, Franceschi S, Melbye M, Boffetta P, Clavel J, Linet MS, Weisenburger DD, Slager SL. Rationale and Design of the International Lymphoma Epidemiology Consortium (InterLymph) Non-Hodgkin Lymphoma Subtypes Project. *J Natl Cancer Inst Monogr*. 2014 Aug;2014(48):1-14. doi: 10.1093/jncimonographs/igu005. PMID: 25174022; PMCID: PMC4155460.

by different subtypes, each with different risk factors, etiologies, clinical features, and response to therapy et cetera. In the category of mature B-cell neoplasms, the WHO classification describes 34 diseases of the B-lymphatic system.<sup>6</sup> The notion mature B cell neoplasms is used to describe various heterogeneous diseases of the B-lymphatic system. This includes entities like Burkitt lymphoma (BL), Chronic Lymphocytic Leukemia (CLL), Diffuse large B-cell lymphoma (DLBCL), Follicular Lymphoma (FL), Hairy cell leukemia (HCL), and variant hairy cell leukemia (HCL-v), High grade B-cell lymphoma (HGBL) with gene rearrangements, Lymphoplasmacytic Lymphoma (LPL)/Morbus Waldenström, Mantle cell lymphoma (MCL), Marginal zone lymphoma (MZL), Monoclonal B-cell lymphocytosis (MBL), Plasma cell myeloma (PCM), Monoclonal Gammopathy of undetermined Significance (MGUS), Prolymphocytic leukemia (B-PLL) and primary CNS lymphoma. Mycosis fungoides/Sézary syndrome (MF/SS) are adult T cell lymphomas.<sup>7</sup> Aggressive lymphomas like precursor B and T cell lymphoblastic leukemia/lymphoma, Burkitt lymphoma, diffuse large B cell lymphoma, and adult T cell leukemia/lymphoma, and certain other peripheral T cell lymphomas have specific B symptoms including weight loss, night sweats, fever and are often deadly within a few weeks if untreated. Depending on various parameters like tumor stage, grade, type of lymphoma, and other factors the treatment of NHL varies greatly. By time, incidence of NHL<sup>8</sup> rose significantly in most Western countries besides of limited data characterising the sub-type-specific incidence of lymphoid neoplasms. A number of studies were launched in order to identify potential causes of and to understand NHL etiology more broadly did not yield the desired results. Still, the etiology of NHL remains poorly understood. Single studies have reported that even certain infectious agents<sup>9</sup> might be related with risk of specific NHL sub-types, among them such as *Helicobacter*<sup>10</sup> *pylori* too. Among other,

<sup>6</sup>Swerdlow SH, Campo E, Pileri SA, Harris NL, Stein H, Siebert R, Advani R, Ghielmini M, Salles GA, Zelenetz AD, Jaffe ES. The 2016 revision of the World Health Organization classification of lymphoid neoplasms. *Blood*. 2016 May 19;127(20):2375-90. doi: 10.1182/blood-2016-01-643569. Epub 2016 Mar 15. PMID: 26980727; PMCID: PMC4874220.

<sup>7</sup>Morton LM, Sampson JN, Cerhan JR, Turner JJ, Vajdic CM, Wang SS, Smedby KE, de Sanjosé S, Monnereau A, Benavente Y, Bracci PM, Chiu BC, Skibola CF, Zhang Y, Mbulaiteye SM, Spriggs M, Robinson D, Norman AD, Kane EV, Spinelli JJ, Kelly JL, La Vecchia C, Dal Maso L, Maynadié M, Kadin ME, Cocco P, Costantini AS, Clarke CA, Roman E, Miligi L, Colt JS, Berndt SI, Mannetje A, de Roos AJ, Krickler A, Nieters A, Franceschi S, Melbye M, Boffetta P, Clavel J, Linet MS, Weisenburger DD, Slager SL. Rationale and Design of the International Lymphoma Epidemiology Consortium (InterLymph) Non-Hodgkin Lymphoma Subtypes Project. *J Natl Cancer Inst Monogr*. 2014 Aug;2014(48):1-14. doi: 10.1093/jncimonographs/igu005. PMID: 25174022; PMCID: PMC4155460.

<sup>8</sup>van Leeuwen MT, Turner JJ, Joske DJ, Falster MO, Srasuebkul P, Meagher NS, Grulich AE, Giles GG, Vajdic CM. Lymphoid neoplasm incidence by WHO subtype in Australia 1982-2006. *Int J Cancer*. 2014 Nov 1;135(9):2146-56. doi: 10.1002/ijc.28849. Epub 2014 Apr 2. PMID: 24639369.

<sup>9</sup>Engels EA. Infectious agents as causes of non-Hodgkin lymphoma. *Cancer Epidemiol Biomarkers Prev*. 2007 Mar;16(3):401-4. doi: 10.1158/1055-9965.EPI-06-1056. Epub 2007 Mar 2. PMID: 17337646.

<sup>10</sup>Suarez F, Lortholary O, Hermine O, Lecuit M. Infection-associated lymphomas derived from marginal zone B cells: a model of antigen-driven lymphoproliferation. *Blood*. 2006 Apr 15;107(8):3034-44. doi: 10.1182/blood-2005-09-3679. Epub 2006 Jan 5. PMID: 16397126.

Mueller et al.<sup>11</sup> found elevated<sup>12, 13, 14, 15, 16</sup> levels of IgG against EBV VCA in NHL populations.

### Epstein–Barr virus

Epstein–Barr virus (EBV), a double-stranded deoxyribonucleic acid (DNA)<sup>17</sup> human  $\gamma$ -herpes virus 4 (HHV4)<sup>18</sup>, with a 170-kb-large genome<sup>19</sup> which encodes for various proteins and non-coding RNAs has been discovered 1964 by Michael Anthony Epstein, Bert Geoffrey Achong and Yvonne M. Barr<sup>20</sup>. After a generally asymptomatic primary EBV infection of mainly B-cells and epithelial cells usually during childhood, EBV resides latently<sup>21</sup> in resting B<sup>22</sup> cells for a lifetime.<sup>23</sup> However, under normal circumstances, an EBV infection is controlled by human immune system and individuals carrying EBV do not suffer from the viral infection. A possible outgrowth of EBV-transformed B lymphocytes in healthy EBV infected individuals is prevented by the presence of intact T lymphocyte-mediated

<sup>11</sup>Mueller N, Mohar A, Evans A, Harris NL, Comstock GW, Jellum E, Magnus K, Orentreich N, Polk BF, Vogelmann J. Epstein-Barr virus antibody patterns preceding the diagnosis of non-Hodgkin's lymphoma. *Int J Cancer*. 1991 Sep 30;49(3):387-93. doi: 10.1002/ijc.2910490313. PMID: 1655660.

<sup>12</sup>Lehtinen T, Lumio J, Dillner J, Hakama M, Knekt P, Lehtinen M, Teppo L, Leinikki P. Increased risk of malignant lymphoma indicated by elevated Epstein-Barr virus antibodies—a prospective study. *Cancer Causes Control*. 1993 May;4(3):187-93. doi: 10.1007/BF00051312. PMID: 8391336.

<sup>13</sup>Rothman N, Cantor KP, Blair A, Bush D, Brock JW, Helzlsouer K, Zahm SH, Needham LL, Pearson GR, Hoover RN, Comstock GW, Strickland PT. A nested case-control study of non-Hodgkin lymphoma and serum organochlorine residues. *Lancet*. 1997 Jul 26;350(9073):240-4. doi: 10.1016/S0140-6736(97)02088-6. PMID: 9242800.

<sup>14</sup>Hardell E, Eriksson M, Lindström G, Van Bavel B, Linde A, Carlberg M, Liljegren G. Case-control study on concentrations of organohalogen compounds and titers of antibodies to Epstein-Barr virus antigens in the etiology of non-Hodgkin lymphoma. *Leuk Lymphoma*. 2001 Aug;42(4):619-29. doi: 10.3109/10428190109099322. PMID: 11697490.

<sup>15</sup>Hardell K, Carlberg M, Hardell L, Björnfoth H, Ericson Jogsten I, Eriksson M, Van Bavel B, Lindström G. Concentrations of organohalogen compounds and titres of antibodies to Epstein-Barr virus antigens and the risk for non-Hodgkin lymphoma. *Oncol Rep*. 2009 Jun;21(6):1567-76. doi: 10.3892/or.00000389. PMID: 19424638.

<sup>16</sup>de Sanjosé S, Bosch R, Schouten T, Verkuijlen S, Nieters A, Foretova L, Maynadié M, Cocco PL, Staines A, Becker N, Brennan P, Benavente Y, Boffetta P, Meijer CJ, Middeldorp JM. Epstein-Barr virus infection and risk of lymphoma: immunoblot analysis of antibody responses against EBV-related proteins in a large series of lymphoma subjects and matched controls. *Int J Cancer*. 2007 Oct 15;121(8):1806-12. doi: 10.1002/ijc.22857. PMID: 17557295.

<sup>17</sup>James Dewey Watson, Francis Harry Compton Crick. Molecular structure of nucleic acids; a structure for deoxyribose nucleic acid. *Nature*. 1953 Apr 25;171(4356):737-8. doi: 10.1038/171737a0. PMID: 13054692.

<sup>18</sup>Walker PJ, Siddell SG, Lefkowitz EJ, Mushegian AR, Adriaenssens EM, Alfenas-Zerbini P, Davison AJ, Dempsey DM, Dutilh BE, García ML, Harrach B, Harrison RL, Hendrickson RC, Junglen S, Knowles NJ, Krupovic M, Kuhn JH, Lambert AJ, Lobočka M, Nibert ML, Oksanen HM, Orton RJ, Robertson DL, Rubino L, Sabanadzovic S, Simmonds P, Smith DB, Suzuki N, Van Doerslaer K, Vandamme AM, Varsani A, Zerbini FM. Changes to virus taxonomy and to the International Code of Virus Classification and Nomenclature ratified by the International Committee on Taxonomy of Viruses (2021). *Arch Virol*. 2021 Sep;166(9):2633-2648. doi: 10.1007/s00705-021-05156-1. PMID: 34231026.

<sup>19</sup>Feederle R, Klinke O, Kutikhin A, Poirey R, Tsai MH, Delecluse HJ. Epstein-Barr Virus: From the Detection of Sequence Polymorphisms to the Recognition of Viral Types. *Curr Top Microbiol Immunol*. 2015;390(Pt 1):119-48. doi: 10.1007/978-3-319-228228\_7. PMID: 26424646.

<sup>20</sup>Michael Anthony Epstein, Bert Geoffrey Achong, Yvonne M. Barr. Virus Particles in Cultured Lymphoblasts from Burkitt's Lymphoma. *Lancet*. 1964 Mar 28;1(7335):702-3. doi: 10.1016/s0140-6736(64)91524-7. PMID: 14107961.

<sup>21</sup>Babcock GJ, Decker LL, Völk M, Thorley-Lawson DA. EBV persistence in memory B cells in vivo. *Immunity*. 1998 Sep;9(3):395-404. doi: 10.1016/s1074-7613(00)80622-6. PMID: 9768759.

<sup>22</sup>Miyashita EM, Yang B, Babcock GJ, Thorley-Lawson DA. Identification of the site of Epstein-Barr virus persistence in vivo as a resting B cell. *J Virol*. 1997 Jul;71(7):4882-91. doi: 10.1128/JVI.71.7.4882-4891.1997. Erratum in: *J Virol* 1998 Nov;72(11):9419. PMID: 9188550; PMCID: PMC191718.

<sup>23</sup>Amon W, Farrell PJ. Reactivation of Epstein-Barr virus from latency. *Rev Med Virol*. 2005 May-Jun;15(3):149-56. doi: 10.1002/rmv.456. PMID: 15546128.

immunity.<sup>24, 25</sup> At the end, up to 95% of the adult population worldwide<sup>26</sup> are infected by EBV (see table 1) at some time during their life span while the EBV seroprevalence increases with age.

**Table 1.** EBV seroprevalence increases with age.

Age y=year	EBV VCA IgG Pos no. (%)	EBV EBNA1 IgG Pos no. (%)	EBV VCA IgM Pos no. (%)	EBV VCA IgA Pos no. (%)	EB EA/D IgA Pos no. (%)
0–5y	283 (66.59)	141 (58.51)	65 (14.57)	52 (14.57)	26 (7.12)
6–10y	431 (84.34)	226 (78.75)	55 (10.24)	93 (22.79)	39 (9.18)
11–20y	784 (92.89)	413 (86.95)	123 (10.41)	178 (23.73)	95 (12.20)
21–30y	809 (98.54)	271 (95.43)	88 (6.25)	192 (26.10)	120 (15.33)
31–40y	853 (98.84)	203 (94.86)	40 (3.06)	219 (22.71)	123 (11.80)
41–50y	892 (99.78)	202 (97.57)	36 (2.76)	282 (22.54)	171 (13.13)
51–60y	957 (99.79)	248 (96.12)	37 (2.62)	301 (27.54)	184 (15.79)
61–101y	902 (99.01)	258 (93.82)	29 (2.03)	258 (33.42)	146 (18.36)

Meanwhile, various methods for the diagnosis of an Epstein-Barr virus (EBV) infection are available. In fact, it is necessary to differentiate<sup>27, 28, 29</sup> between a primary EBV infection and an EBV reactivation. Serological tests for immunoglobulin G (IgG)- and immunoglobulin M (IgM)-antibodies to Epstein-Barr virus viral capsid antigen (VCA) and IgG-antibodies to Epstein-Barr nuclear antigen 1 (EBNA-1) are frequently used to define infection status and for the differential diagnosis too. The presence of EBV VCA IgG and EBV VCA IgM without EBV EBNA-1 IgG indicates more or less an acute EBV infection, whereas the presence of VCA IgG and EBNA-1 IgG without VCA IgM is typical of past EBV infection<sup>30</sup>. However, immunoglobulin G (IgG) is representing approximately 75% of serum antibodies in humans and is subject to very specific pharmacokinetics<sup>31</sup> and clearance. The plasma half-life<sup>32</sup> of IgG<sup>33, 34, 35</sup> is about 21 day. The human immune system does not always

<sup>24</sup>Khanna R, Burrows SR. Role of cytotoxic T lymphocytes in Epstein-Barr virus-associated diseases. *Annu Rev Microbiol.* 2000;54:19-48. doi: 10.1146/annurev.micro.54.1.19. PMID: 11018123.

<sup>25</sup>Thorley-Lawson DA, Gross A. Persistence of the Epstein-Barr virus and the origins of associated lymphomas. *N Engl J Med.* 2004 Mar 25;350(13):1328-37. doi: 10.1056/NEJMr032015. PMID: 15044644.

<sup>26</sup>Cui J, Yan W, Xu S, Wang Q, Zhang W, Liu W, Ni A. Anti-Epstein-Barr virus antibodies in Beijing during 2013-2017: What we have found in the different patients. *PLoS One.* 2018 Mar 1;13(3):e0193171. doi: 10.1371/journal.pone.0193171. PMID: 29494658; PMCID: PMC5832223.

<sup>27</sup>Robertson P, Beynon S, Whybin R, Brennan C, Vollmer-Conna U, Hickie I, Lloyd A. Measurement of EBV-IgG anti-VCA avidity aids the early and reliable diagnosis of primary EBV infection. *J Med Virol.* 2003 Aug;70(4):617-23. doi: 10.1002/jmv.10439. PMID: 12794726.

<sup>28</sup>De Paschale M, Clerici P. Serological diagnosis of Epstein-Barr virus infection: Problems and solutions. *World J Virol.* 2012 Feb 12;1(1):31-43. doi: 10.5501/wjv.v1.i1.31. PMID: 24175209; PMCID: PMC3782265.

<sup>29</sup>De Paschale M, Agrappi C, Manco MT, Mirri P, Viganò EF, Clerici P. Seroepidemiology of EBV and interpretation of the "isolated VCA IgG" pattern. *J Med Virol.* 2009 Feb;81(2):325-31. doi: 10.1002/jmv.21373. PMID: 19107979.

<sup>30</sup>De Paschale M, Clerici P. Serological diagnosis of Epstein-Barr virus infection: Problems and solutions. *World J Virol.* 2012 Feb 12;1(1):31-43. doi: 10.5501/wjv.v1.i1.31. PMID: 24175209; PMCID: PMC3782265.

<sup>31</sup>WALDMANN TA, SCHWAB PJ. IGG (7 S GAMMA GLOBULIN) METABOLISM IN HYPOGAMMAGLOBULINEMIA: STUDIES IN PATIENTS WITH DEFECTIVE GAMMA GLOBULIN SYNTHESIS, GASTROINTESTINAL PROTEIN LOSS, OR BOTH. *J Clin Invest.* 1965 Sep;44(9):1523-33. doi: 10.1172/JCI105259. PMID: 14332165; PMCID: PMC292634.

<sup>32</sup>GORDON EB, WIENER AS. Studies on human serum gamma globulin. I. Half-life and rate of production. *J Lab Clin Med.* 1957 Feb;49(2):258-62. PMID: 13398691.

<sup>33</sup>Morell A, Terry WD, Waldmann TA. Metabolic properties of IgG subclasses in man. *J Clin Invest.* 1970 Apr;49(4):673-80. doi: 10.1172/JCI106279. PMID: 5443170; PMCID: PMC322522.

<sup>34</sup>Mankarious S, Lee M, Fischer S, Pyun KH, Ochs HD, Oxelius VA, Wedgwood RJ. The half-lives of IgG subclasses and specific antibodies in patients with primary immunodeficiency who are receiving intravenously administered immunoglobulin. *J Lab Clin Med.* 1988 Nov;112(5):634-40. PMID: 3183495.

<sup>35</sup>Bonilla FA. Pharmacokinetics of immunoglobulin administered via intravenous or subcutaneous routes. *Immunol Allergy Clin*

posses a reason or a possibility to produce EBV IgG or IgM antibodies. Especially, if there is no EBV re/infection, IgG is reduced by half about every 21 days and used for other purposes. Thus far, false positive and false negative IgG based results are theoretically possible. Furthermore, the sensitivity and the specificity of EBV tests is not always equal to 100 %. At the end, serological tests for antibodies specific for Epstein-Barr virus (EBV) antigens are of use to identify the EBV infection status of a single person.<sup>36</sup> However, the specific tests for anti-EBV antibodies use different antigens or substrates or and even various technologies. Therefore, depending upon specificity and sensitivity of a test<sup>37, 38, 39</sup> used, serological findings may sometimes be difficult to rely upon. EBV is discussed as the etiologic agent of infectious mononucleosis (Pfeiffersches Drüsenfieber, Morbus Pfeiffer),<sup>40, 41</sup>, EBV DNA was detected in tissues from patients with nasopharyngeal carcinoma<sup>42, 43, 44</sup> and other tissues too. EBV is the cause of multiple sclerosis<sup>45, 46, 47, 48, 49, 50</sup>, of rheumatoid arthritis

North Am. 2008 Nov;28(4):803-19, ix. doi: 10.1016/j.jac.2008.06.006. PMID: 18940575.

<sup>36</sup>De Paschale M, Clerici P. Serological diagnosis of Epstein-Barr virus infection: Problems and solutions. *World J Virol.* 2012 Feb 12;1(1):31-43. doi: 10.5501/wjv.v1.i1.31. PMID: 24175209; PMCID: PMC3782265.

<sup>37</sup>Gärtner BC, Hess RD, Bandt D, Kruse A, Rethwilm A, Roemer K, Mueller-Lantsch N. Evaluation of four commercially available Epstein-Barr virus enzyme immunoassays with an immunofluorescence assay as the reference method. *Clin Diagn Lab Immunol.* 2003 Jan;10(1):78-82. doi: 10.1128/cdli.10.1.78-82.2003. PMID: 12522043; PMCID: PMC145280.

<sup>38</sup>Bruu AL, Hjetland R, Holter E, Mortensen L, Nats O, Petterson W, Skar AG, Skarpaas T, Tjade T, Asjø B. Evaluation of 12 commercial tests for detection of Epstein-Barr virus-specific and heterophile antibodies. *Clin Diagn Lab Immunol.* 2000 May;7(3):451-6. doi: 10.1128/CDLI.7.3.451-456.2000. PMID: 10799460; PMCID: PMC95893.

<sup>39</sup>de Ory F, Guisasaola ME, Sanz JC, García-Bermejo I. Evaluation of four commercial systems for the diagnosis of Epstein-Barr virus primary infections. *Clin Vaccine Immunol.* 2011 Mar;18(3):444-8. doi: 10.1128/CVI.00486-10. Epub 2010 Dec 29. PMID: 21191077; PMCID: PMC3067387.

<sup>40</sup>Graser F. Hundert Jahre Pfeiffersches Drüsenfieber [100 years of Pfeiffer's glandular fever, Article in German]. *Klin Padiatr.* 1991 May-Jun;203(3):187-90. German. doi: 10.1055/s-2007-1025428. PMID: 1857056.

<sup>41</sup>Henle G, Henle W, Diehl V. Relation of Burkitt's tumor-associated herpes-ytpye virus to infectious mononucleosis. *Proc Natl Acad Sci U S A.* 1968 Jan;59(1):94-101. doi: 10.1073/pnas.59.1.94. PMID: 5242134; PMCID: PMC286007.

<sup>42</sup>zur Hausen H, Schulte-Holthausen H, Klein G, Henle W, Henle G, Clifford P, Santesson L. EBV DNA in biopsies of Burkitt tumours and anaplastic carcinomas of the nasopharynx. *Nature.* 1970 Dec 12;228(5276):1056-8. doi: 10.1038/2281056a0. PMID: 4320657.

<sup>43</sup>Gunvén P, Klein G, Henle G, Henle W, Clifford P. Epstein-Barr virus in Burkitt's lymphoma and nasopharyngeal carcinoma. Antibodies to EBV associated membrane and viral capsid antigens in Burkitt lymphoma patients. *Nature.* 1970 Dec 12;228(5276):1053-6. doi: 10.1038/2281053a0. PMID: 4320656.

<sup>44</sup>Barukčić, Ilija. (2022). Without Epstein-Barr virus infection, no nasopharyngeal carcinoma. *Causation*, 17(4), 5–65. <https://doi.org/10.5281/zenodo.6386619>

<sup>45</sup>Nikoskelainen J, Panelius M, Salmi A. E.B. virus and multiple sclerosis. *Br Med J.* 1972 Oct 14;4(5832):111. doi: 10.1136/bmj.4.5832.111. PMID: 4342670; PMCID: PMC1786242.

<sup>46</sup>Barukčić, K. and Barukčić, I. (2016) Epstein Barr Virus—The Cause of Multiple Sclerosis. *Journal of Applied Mathematics and Physics*, 4, 1042-1053. doi: 10.4236/jamp.2016.46109.

<sup>47</sup>Barukčić, Ilija. (2018). Epstein-Barr virus is the cause of multiple sclerosis. *INTERNATIONAL JOURNAL OF CURRENT MEDICAL AND PHARMACEUTICAL RESEARCH*. Volume 4; Issue 9(A); September 2018; Page No. 3674-3682 <https://doi.org/10.5281/zenodo.3943315>

<sup>48</sup>Bjornevik K, Cortese M, Healy BC, Kuhle J, Mina MJ, Leng Y, Elledge SJ, Niebuhr DW, Scher AI, Munger KL, Ascherio A. Longitudinal analysis reveals high prevalence of Epstein-Barr virus associated with multiple sclerosis. *Science.* 2022 Jan 21;375(6578):296-301. doi: 10.1126/science.abj8222. Epub 2022 Jan 13. PMID: 35025605.

<sup>49</sup>Bjornevik K, Cortese M, Healy BC, Kuhle J, Mina MJ, Leng Y, Elledge SJ, Niebuhr DW, Scher AI, Munger KL, Ascherio A. Longitudinal analysis reveals high prevalence of Epstein-Barr virus associated with multiple sclerosis. *Science.* 2022 Jan 21;375(6578):296-301. doi: 10.1126/science.abj8222. Epub 2022 Jan 13. PMID: 35025605.

<sup>50</sup>Ludvig M. Sollid, Epstein-Barr virus as a driver of multiple sclerosis, *Science Immunology*, 7, 70, (2022). </doi/10.1126/sciimmunol.abo7799>

<sup>51</sup> et cetera. Among other, high dose intravenous(i.v.) <sup>52</sup> , <sup>53</sup> L-ascorbic acid (vitamin C) <sup>54</sup> , <sup>55</sup> , <sup>56</sup> , <sup>57</sup> , <sup>58</sup> , valacyclovir <sup>59</sup> , <sup>60</sup> or prednisolon <sup>61</sup> , <sup>62</sup> have been used to treat EBV. It has been reported that anti-EBNA1 EBV antibody levels decreased <sup>63</sup> , <sup>64</sup> by a supplementation with high-dose oral 25-hydroxyvitamin D3 (25(OH)D3). Regrettably, despite the massive EBV caused damage to individual <sup>65</sup> human beings and the hole human society, there is no antiviral drug approved for the treatment of (chronic active) EBV infections. <sup>66</sup>

Unfortunately, other studies found contradictory results with respect to the relationship between

<sup>51</sup>Katarina Barukčić, Jan Pavo Barukčić, Ilija Barukčić. Epstein-Barr virus is the cause of rheumatoid arthritis. ROMANIAN JOURNAL OF RHEUMATOLOGY – VOLUME 27, NO. 4, 2018. 148-163.

<sup>52</sup>Riordan HD, Hunninghake RB, Riordan NH, Jackson JJ, Meng X, Taylor P, Casciari JJ, González MJ, Miranda-Massari JR, Mora EM, Rosario N, Rivera A. Intravenous ascorbic acid: protocol for its application and use. P R Health Sci J. 2003 Sep;22(3):287-90. PMID: 14619456.

<sup>53</sup>Shatzer AN, Espey MG, Chavez M, Tu H, Levine M, Cohen JI. Ascorbic acid kills Epstein-Barr virus positive Burkitt lymphoma cells and Epstein-Barr virus transformed B-cells in vitro, but not in vivo. Leuk Lymphoma. 2013 May;54(5):1069-78. doi: 10.3109/10428194.2012.739686. Epub 2012 Nov 15. PMID: 23067008; PMCID: PMC4055524.

<sup>54</sup>Lind, James (1716-1794). A treatise of the scurvy, in three parts. Containing an inquiry into the nature, causes, and cure, of that disease. Together with a critical and chronological view of what has been published on the subject. Edinburgh (Scotland) : Printed by Sands, Murray and Cochran, for A. Kincaid & A. Donsaldson. 1753. 456 pages.

<sup>55</sup>Svirbely JL, Szent-Györgyi A. The chemical nature of vitamin C. Biochem J. 1932;26(3):865-70. doi: 10.1042/bj0260865. PMID: 16744896; PMCID: PMC1260981.

<sup>56</sup>Svirbely JL, Szent-Györgyi A. The chemical nature of vitamin C. Biochem J. 1933;27(1):279-85. PMID: 16745082; PMCID: PMC1252872.

<sup>57</sup>Linus Pauling. THE NATURE OF THE CHEMICAL BOND. APPLICATION OF RESULTS OBTAINED FROM THE QUANTUM MECHANICS AND FROM A THEORY OF PARAMAGNETIC SUSCEPTIBILITY TO THE STRUCTURE OF MOLECULES. Journal of the American Chemical Society 1931, 53, 4, 1367-1400.

<sup>58</sup>King CG, Waugh WA. THE CHEMICAL NATURE OF VITAMIN C. Science. 1932 Apr 1;75(1944):357-8. doi: 10.1126/science.75.1944.357-a. PMID: 17750032.

<sup>59</sup>Lerner AM, Beqaj SH, Deeter RG, Dworkin HJ, Zervos M, Chang CH, Fitzgerald JT, Goldstein J, O'Neill W. A six-month trial of valacyclovir in the Epstein-Barr virus subset of chronic fatigue syndrome: improvement in left ventricular function. Drugs Today (Barc). 2002 Aug;38(8):549-61. doi: 10.1358/dot.2002.38.8.820095. PMID: 12582420.

<sup>60</sup>Lerner AM, Beqaj SH, Deeter RG, Fitzgerald JT. Valacyclovir treatment in Epstein-Barr virus subset chronic fatigue syndrome: thirty-six months follow-up. In Vivo. 2007 Sep-Oct;21(5):707-13. PMID: 18019402.

<sup>61</sup>Sawada A, Inoue M, Kawa K. How we treat chronic active Epstein-Barr virus infection. Int J Hematol. 2017 Apr;105(4):406-418. doi: 10.1007/s12185-017-2192-6. Epub 2017 Feb 16. PMID: 28210942.

<sup>62</sup>Tynell E, Aurelius E, Brandell A, Julander I, Wood M, Yao QY, Rickinson A, Akerlund B, Andersson J. Acyclovir and prednisolone treatment of acute infectious mononucleosis: a multicenter, double-blind, placebo-controlled study. J Infect Dis. 1996 Aug;174(2):324-31. doi: 10.1093/infdis/174.2.324. PMID: 8699062.

<sup>63</sup>Røsjø E, Lossius A, Abdelmagid N, Lindstrøm JC, Kampman MT, Jørgensen L, Sundstrøm P, Olsson T, Steffensen LH, Torkildsen Ø, Holmøy T. Effect of high-dose vitamin D3 supplementation on antibody responses against Epstein-Barr virus in relapsing-remitting multiple sclerosis. Mult Scler. 2017 Mar;23(3):395-402. doi: 10.1177/1352458516654310. Epub 2016 Jul 11. PMID: 27325604.

<sup>64</sup>Najafipoor A, Roghanian R, Zarkesh-Esfahani SH, Bouzari M, Etemadifar M. The beneficial effects of vitamin D3 on reducing antibody titers against Epstein-Barr virus in multiple sclerosis patients. Cell Immunol. 2015 Mar;294(1):9-12. doi: 10.1016/j.cellimm.2015.01.009. Epub 2015 Jan 28. PMID: 25666504.

<sup>65</sup>Biebl A, Webersinke C, Traxler B, Povysil B, Furthner D, Schmitt K, Weis S. Fatal Epstein-Barr virus encephalitis in a 12-year-old child: an underappreciated neurological complication? Nat Clin Pract Neurol. 2009 Mar;5(3):171-4. doi: 10.1038/ncpneu1043. PMID: 19262593.

<sup>66</sup>Andrei G, Trompet E, Snoeck R. Novel Therapeutics for EpsteinBarr Virus. Molecules. 2019 Mar 12;24(5):997. doi: 10.3390/molecules24050997. PMID: 30871092; PMCID: PMC6429425.

EBV and NHL. <sup>67</sup>, <sup>68</sup>, <sup>69</sup>, <sup>70</sup> Even today, the etiology of NHL <sup>71</sup>, <sup>72</sup> remains an open <sup>73</sup>, <sup>74</sup> question.

<sup>67</sup>Cohen JI. Epstein-Barr virus infection. *N Engl J Med*. 2000 Aug 17;343(7):481-92. doi: 10.1056/NEJM200008173430707. PMID: 10944566.

<sup>68</sup>Leong IT, Fernandes BJ, Mock D. Epstein-Barr virus detection in non-Hodgkin's lymphoma of the oral cavity: an immunocytochemical and in situ hybridization study. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 2001 Aug;92(2):184-93. doi: 10.1067/moe.2001.116155. PMID: 11505266.

<sup>69</sup>De Roos AJ, Martínez-Maza O, Jerome KR, Mirick DK, Kopecky KJ, Madeleine MM, Magpantay L, Edlefsen KL, Lacroix AZ. Investigation of Epstein-Barr virus as a potential cause of B-cell non-Hodgkin lymphoma in a prospective cohort. *Cancer Epidemiol Biomarkers Prev*. 2013 Oct;22(10):1747-55. doi: 10.1158/1055-9965.EPI-13-0240. Epub 2013 Jul 24. PMID: 23885038; PMCID: PMC4193346.

<sup>70</sup>Thorley-Lawson DA, Gross A. Persistence of the Epstein-Barr virus and the origins of associated lymphomas. *N Engl J Med*. 2004 Mar 25;350(13):1328-37. doi: 10.1056/NEJMra032015. PMID: 15044644.

<sup>71</sup>Vaillant V, Reiter A, Zimmermann M, Wagner HJ. Seroepidemiological analysis and literature review of the prevalence of Epstein-Barr virus and herpesvirus infections in pediatric cases with non-Hodgkin lymphoma in Central Europe. *Pediatr Blood Cancer*. 2019 Jul;66(7):e27752. doi: 10.1002/pbc.27752. Epub 2019 Apr 12. PMID: 30977593.

<sup>72</sup>Orem J, Sandin S, Mbidde E, Mangan FW, Middeldorp J, Weiderpass E. Epstein-Barr virus viral load and serology in childhood non-Hodgkin's lymphoma and chronic inflammatory conditions in Uganda: implications for disease risk and characteristics. *J Med Virol*. 2014 Oct;86(10):1796-803. doi: 10.1002/jmv.23988. Epub 2014 Jun 2. PMID: 24889739.

<sup>73</sup>Qin C, Huang Y, Feng Y, Li M, Guo N, Rao H. Clinicopathological features and EBV infection status of lymphoma in children and adolescents in South China: a retrospective study of 662 cases. *Diagn Pathol*. 2018 Feb 27;13(1):17. doi: 10.1186/s13000-018-0693-0. PMID: 29482573; PMCID: PMC5828429.

<sup>74</sup>Bar



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## 2. Material and methods

Scientific knowledge and objective reality are more than only interrelated. It cannot be repeated often enough that objective reality or processes of objective reality is the foundation of any scientific knowledge. Our human experience teaches us however that seen by light, grey is never merely simply grey, and looked at from different angles, many paths may lead to climb up a certain mountain. In general, it is appropriate to ensure as much as possible a broader consideration of a research question and to take into account the different facets and viewpoints of an issue investigated in order to reach a goal.

### 2.1. Material

#### 2.1.1. Search strategy for identification of studies

The electronic database PubMed<sup>75</sup> was searched for suitable articles and yielded 167 results. The identified articles were re-analysed, reporting followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses<sup>76, 77</sup> (PRISMA) guidelines.

#### 2.1.2. The study of Kimberly A Bertrand et al., 2010

Kimberly A Bertrand et al.<sup>78</sup> conducted a case-control study (with 340 cases and 662 matched controls) of apparently immunocompetent men and women nested in the Nurses' Health Study and the Physicians' Health Study cohorts in order to evaluate whether Epstein-Barr virus infection is related NHL. Bertrand et al. reported that "... the final study population consisted of 340 NHL cases and 662 controls. Of these, 319 cases (94%) and 629 controls (95%) were EBV seropositive. "More or less, Bertrand et al. concluded that no evidence has been found that EBV is related to NHL.

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<sup>75</sup>PubMed: Epstein-Barr virus and IgG and non Hodgkin lymphoma

<sup>76</sup>Moher D, Liberati A, Tetzlaff J, Altman DG; PRISMA Group. Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement. *PLoS Med.* 2009 Jul 21;6(7):e1000097. doi: 10.1371/journal.pmed.1000097. Epub 2009 Jul 21. PMID: 19621072; PMCID: PMC2707599.

<sup>77</sup>Liberati A, Altman DG, Tetzlaff J, Mulrow C, Gøtzsche PC, Ioannidis JP, Clarke M, Devereaux PJ, Kleijnen J, Moher D. The PRISMA statement for reporting systematic reviews and meta-analyses of studies that evaluate healthcare interventions: explanation and elaboration. *BMJ.* 2009 Jul 21;339:b2700. doi: 10.1136/bmj.b2700. PMID: 19622552; PMCID: PMC2714672.

<sup>78</sup>Bertrand KA, Birmann BM, Chang ET, Spiegelman D, Aster JC, Zhang SM, Laden F. A prospective study of Epstein-Barr virus antibodies and risk of non-Hodgkin lymphoma. *Blood.* 2010 Nov 4;116(18):3547-53. doi: 10.1182/blood-2010-05-282715. Epub 2010 Jul 20. PMID: 20647565; PMCID: PMC2981477.

### 2.1.3. The study of Kabyemera et al., 2013

The incidence of lymphomas varies. About 60% of all childhood lymphomas are classified as NHL.<sup>79</sup> Non-Hodgkin's Lymphomas (NHL) are common in African children too. Kabyemera et al.<sup>81</sup> conducted a matched case control study of NHL subtypes in north-western Tanzania. Peripheral blood samples in children under 15 years of age were collected and EBV DNA levels were estimated by multiplex real-time PCR. Kabyemera et al. found that "NHLs are ... are strongly associated with EBV load in peripheral blood."<sup>82</sup>

### 2.1.4. The study of Anneclaire J De Roos et al., 2013

Anneclaire J De Roos et al.<sup>83</sup> were of the opinion that poor control of Epstein-Barr virus (EBV) infection, leading to reactivation of the virus might end up at non-Hodgkin lymphoma. A case-control study nested within the Women's Health Initiative Observational Study cohort was conducted and found 454 individuals with EBV IgG in 491 B-cell NHL cases and 446 individuals with EBV IgG in 491 controls. De Roos et al. concluded that EBV is not playing a causal role in B-cell NHL in general population women.

### 2.1.5. The study of Teras et al., 2015

Lauren R Teras et al.<sup>84</sup> investigated the relationship between plasma EBV antibodies and NHL in the Cancer Prevention Study-II (CPS-II) Nutrition Cohort. At the end, the CPS-II study included 225 NHL cases and 2:1 matched controls.

### 2.1.6. Statistical methods

The probability of the exclusion (Barukčić, 2021c) relationship(see also Barukčić, 2021a)  $p(\text{EXCL})$  has been calculated and tested for statistical significance. The chi-square goodness of fit

<sup>79</sup>Sandlund JT, Downing JR, Crist WM. Non-Hodgkin's lymphoma in childhood. *N Engl J Med*. 1996 May 9;334(19):1238-48. doi: 10.1056/NEJM199605093341906. PMID: 8606720.

<sup>80</sup>Young JL Jr, Ries LG, Silverberg E, Horm JW, Miller RW. Cancer incidence, survival, and mortality for children younger than age 15 years. *Cancer*. 1986 Jul 15;58(2 Suppl):598-602. doi: 10.1002/1097-0142(19860715)58:2+;598::aid-cnrcr2820581332j3.0.co;2-c. PMID: 3719551.

<sup>81</sup>Kabyemera R, Masalu N, Rambau P, Kamugisha E, Kidenya B, De Rossi A, Petrara MR, Mwizamuholya D. Relationship between non-Hodgkin's lymphoma and blood levels of Epstein-Barr virus in children in north-western Tanzania: a case control study. *BMC Pediatr*. 2013 Jan 7;13:4. doi: 10.1186/1471-2431-13-4. PMID: 23294539; PMCID: PMC3547779.

<sup>82</sup>Ibid.

<sup>83</sup>De Roos AJ, Martínez-Maza O, Jerome KR, Mirick DK, Kopecky KJ, Madeleine MM, Magpantay L, Edlefsen KL, Lacroix AZ. Investigation of Epstein-Barr virus as a potential cause of B-cell non-Hodgkin lymphoma in a prospective cohort. *Cancer Epidemiol Biomarkers Prev*. 2013 Oct;22(10):1747-55. doi: 10.1158/1055-9965.EPI-13-0240. Epub 2013 Jul 24. PMID: 23885038; PMCID: PMC4193346. Format:

<sup>84</sup>Teras LR, Rollison DE, Pawlita M, Michel A, Brozy J, de Sanjose S, Blase JL, Gapstur SM. Epstein-Barr virus and risk of non-Hodgkin lymphoma in the cancer prevention study-II and a meta-analysis of serologic studies. *Int J Cancer*. 2015 Jan 1;136(1):108-16. doi: 10.1002/ijc.28971. Epub 2014 Jun 5. PMID: 24831943. see also: <https://pubmed.ncbi.nlm.nih.gov/24831943/>

test with one degree of freedom has been used to test whether the sample data published fit a certain theoretical distribution in the population. Additionally, the P Value has been calculated approximately (see also Barukčić, 2019e). The causal relationship  $k$  (Barukčić, 2016b, 2020a, 2021c) has been calculated to evaluate a possible causal relationship between the events. The hyper-geometric (Fisher, 1922, Gonin, 1936, Huygens and van Schooten, 1657, Pearson, 1899) distribution (HGD) has been used to test the one-sided significance of the causal relationship  $k$ . Bringing different studies together for analysing them or doing a meta-analysis is not without problems. Due to several reasons, there is variability in the data of the studies and there will be differences found. Usually, the heterogeneity among the studies is assessed through  $I^2$  statistics<sup>85</sup>,<sup>86</sup>,<sup>87</sup>. Under usual circumstances, an  $I^2$  value of 25%, 50% and 75% are regarded as low, moderate and high heterogeneity<sup>88</sup>. In this publication, the study (design) bias and the heterogeneity among the studies has been controlled by IOI, the index of independence (Barukčić, 2019c) and IOU, the index of unfairness (Barukčić, 2019d). All the data were analysed using MS Excel (Microsoft Corporation, USA).

P values less than 0.05 were considered statistically significant.

## 2.2. Methods

Definitions should help us to provide and assure a systematic approach to a scientific issue. It also goes without the need of further saying that a definition as such need to be logically consistent and correct.

### 2.2.1. Random variables

As highlighted especially by Albert Einstein (1879-1955) and his coworkers Boris Yakovlevich Podolsky (1896-1966) and Nathan Rosen (1909-1995), "... objective reality ... is independent of any theory ..."<sup>89</sup> (see Einstein et al., 1935, p. 777), objective reality is independent of any observer and of any perceiving subject, objective reality is independent of any measurements. Let us carry this point to epistemological extremes, objective reality is existing independently and outside of human mind and consciousness. However, in its own self-sameness objective reality is different from a random variable too and self-contradictory. Nonetheless, in its difference, in its own contradiction, a random variable itself is self-identical and is in its own self a transition of itself into the other of itself and vice versa. Lastly, a random variable as such is in its own self the opposite of itself. More or less, a random variable is in its own self the unity of identity and difference and finds its own completion in the determinate relationship of self-identity and difference. A random variable as such is in its own

<sup>85</sup>Cochran WG. The combination of estimates from different experiments. *Biometrics* 1954; 10(1): 101-29.

<sup>86</sup>Higgins JP, Thompson SG. Quantifying heterogeneity in a meta-analysis. *Stat Med.* 2002 Jun 15;21(11):1539-58. doi: 10.1002/sim.1186. PMID: 12111919.

<sup>87</sup>Higgins JP, Thompson SG, Deeks JJ, Altman DG. Measuring inconsistency in meta-analyses. *BMJ.* 2003 Sep 6;327(7414):557-60. doi: 10.1136/bmj.327.7414.557. PMID: 12958120; PMCID: PMC192859.

<sup>88</sup>Higgins JP, Thompson SG, Deeks JJ, Altman DG. Measuring inconsistency in meta-analyses. *BMJ.* 2003 Sep 6;327(7414):557-60. doi: 10.1136/bmj.327.7414.557. PMID: 12958120; PMCID: PMC192859.

<sup>89</sup>Einstein, A; B Podolsky; N Rosen (1935-05-15). "Can Quantum-Mechanical Description of Physical Reality be Considered Complete?" (PDF). *Physical Review.* 47 (10): 777–780. DOI:<https://doi.org/10.1103/PhysRev.47.777>

self self-identical and different. This has at least a twofold aspect, identity and difference constitute the determinations of a random variable itself. These two moments of a random variable which are merely different in one and the same identity are constituting as moments of difference the determinations of an opposition too. A self-identical and a different constitute equally the interior nature of itself in relation to one another. The self-identical, determined with reference to an otherness, has within itself the reference-to-other which is the determinateness of the self-identical itself. The difference contains within itself the reference to its non-being, to identity, and vice versa. Identity contains within itself the reference to its non-being, to difference. However, a random variable as such is itself and its other and the identity of difference with itself is at the end a self-reference too. Consequently, a random variable as such has its own determinateness not in an other, but in its own self, it is self-referred, while the reference to its other manifests itself as a self-reference. The other of itself which a random variable as such contains is also the non-being of that in which it is supposed to be contained only as a moment. A random variable as such therefore is, only in so far as its non-being is, and is in an identical relationship with it. The moments of a random variable are different in one and the same identity and as moments of difference are constituting the determinations of an opposition. Closer consideration shows that a random variable as such is only in so far as the same contains a reference to its non-being, to its own other moment (i.e. local hidden variable). A self-identical which is equally a different too is thus far determining an opposition as such. While the one is not as yet self-identical, the other is not as yet different. However, both are different to one another. Nonetheless, the indifference of a random variable as such towards another random variable distinguished from the same has no influence on the fact that a random variable as such is in its own self the unity of identity and difference. At the end, a random variable as such is, only in so far as the other is; it is what it is, through the other, through its own non-being. A random variable is, in so far as the other is not; it is what it is, through the non-being of its own other.

The notion something is widely taken for granted as a foundation of axioms, theorems and theories. But, very broadly put, there are many different kinds of very concrete, single entities with real world implications. Thus far, what is something, what is its own other? In the most general way, there are circumstances where something and its own other existing independently and outside of human mind and consciousness is described mathematically by the notion random variable. Let a **random variable** (Gosset, 1914)  $X$  denote something like a function defined on a probability space, which itself maps from the sample space (Neyman and Pearson, 1933) to the real numbers.

### 2.2.1.1. The Expectation of a Random Variable

**Definition 2.1 (The First Moment Expectation of a Random Variable).** *Summaries of an entire distribution of a random variable (see Kolmogorov, Andreï Nikolaevich, 1950, p. 22 )  $X$ , such as the expected value, or average value, are useful in order to identify where  $X$  is expected to be without describing the entire distribution. For practical and other reasons, we shall limit ourselves here to discrete random variables, while the basic properties of the expectation value of a random variable  $X$  will not be investigated. Thus far, let  $X$  be a discrete random variable with the probability  $p(X)$ . The relationship between the first moment expectation value (see Huygens and van Schooten, 1657, Kolmogorov, Andreï Nikolaevich, 1950, LaPlace, 1812, Whitworth, 1901) of  $X$ , denoted by  $E(X)$ , and*

the probability  $p(X)$ , is given by the equation:

$$\begin{aligned} E(X) &\equiv X \times p(X) \\ &\equiv \Psi(X) \times X \times \Psi^*(X) \end{aligned} \quad (1)$$

where  $\Psi(X)$  is the wave-function (see [Born, 1926](#), [Schrödinger, Erwin Rudolf Josef Alexander, 1926](#)) of  $X$ ,  $\Psi^*(X)$  is the complex conjugate wave-function of  $X$ . Under conditions where  $p(X) \equiv +1$  equation 1 (see p. 17) becomes

$$E(X) \equiv X \quad (2)$$

but not general. The first moment expectation value squared of a random variable  $X$  follows as

$$\begin{aligned} E(X)^2 &\equiv p(X) \times X \times p(X) \times X \\ &\equiv p(X) \times p(X) \times X \times X \\ &\equiv (p(X) \times X)^2 \\ &\equiv E(X) \times E(X) \end{aligned} \quad (3)$$

The ongoing progress with artificial intelligence has the potential to transform human society far beyond any imaginable border of human recognition and can help even to solve problems that otherwise would not be tractable. No wonder, scientist and systems are confronted with large volumes of data (big data) of various natures and from different sources. The use of tensor technology can simplify and accelerate Big data analysis. In other words, let  $X_{kl\mu\nu\dots}$  denote an  $n$ -th index co-variant tensor with the probability  $p(X_{kl\mu\nu\dots})$ . The first moment expectation value (see [Huygens and van Schooten, 1657](#), [Kolmogorov, Andreĭ Nikolaevich, 1950](#), [LaPlace, 1812](#), [Whitworth, 1901](#)) of  $X_{kl\mu\nu\dots}$ , denoted by  $E(X_{kl\mu\nu\dots})$ , is a number defined as follows:

$$E(X_{kl\mu\nu\dots}) \equiv p(X_{kl\mu\nu\dots}) \times X_{kl\mu\nu\dots} \equiv p(X_{kl\mu\nu\dots}) \cap X_{kl\mu\nu\dots} \quad (4)$$

while  $\times$  or  $\cap$  might denote the commutative multiplications of tensors. The first moment expectation value squared of a random variable  $X$  follows as

$$\begin{aligned} {}^2E(X_{kl\mu\nu\dots}) &\equiv p(X_{kl\mu\nu\dots}) \times X_{kl\mu\nu\dots} \times p(X_{kl\mu\nu\dots}) \times X_{kl\mu\nu\dots} \\ &\equiv p(X_{kl\mu\nu\dots}) \times p(X_{kl\mu\nu\dots}) \times X_{kl\mu\nu\dots} \times X_{kl\mu\nu\dots} \\ &\equiv {}^2(p(X_{kl\mu\nu\dots}) \times X_{kl\mu\nu\dots}) \\ &\equiv E(X_{kl\mu\nu\dots}) \times E(X_{kl\mu\nu\dots}) \end{aligned} \quad (5)$$

**Definition 2.2 (The Second Moment Expectation of a Random Variable).** *The second (see [Kolmogorov, Andreĭ Nikolaevich, 1950](#), p. 42) moment expectation value (or more or less arithmetic mean) of a (large) number of independent realizations of a random variable  $X$  follows as:*

$$\begin{aligned} E(X^2) &\equiv p(X) \times X^2 \\ &\equiv (p(X) \times X) \times X \\ &\equiv E(X) \times X \\ &\equiv X \times E(X) \end{aligned} \quad (6)$$

From the point of view of tensor algebra it is

$$\begin{aligned}
 E\left({}^2X_{kl\mu\nu\dots}\right) &\equiv p\left(X_{kl\mu\nu\dots}\right) \times {}^2X_{kl\mu\nu\dots} \\
 &\equiv \left(p\left(X_{kl\mu\nu\dots}\right) \times X_{kl\mu\nu\dots}\right) \times X_{kl\mu\nu\dots} \\
 &\equiv E\left(X_{kl\mu\nu\dots}\right) \times X_{kl\mu\nu\dots} \\
 &\equiv X_{kl\mu\nu\dots} \times E\left(X_{kl\mu\nu\dots}\right)
 \end{aligned}
 \tag{7}$$

**Definition 2.3 (The n-th Moment Expectation of a Random Variable).** *The n-th (see Barukčić, 2020a, 2021c) moment expectation value of a (large) number of independent realizations of a random variable X follows as:*

$$\begin{aligned}
 E\left(X^n\right) &\equiv p\left(X\right) \times X^n \\
 &\equiv \left(p\left(X\right) \times X\right) \times X^{n-1} \\
 &\equiv E\left(X\right) \times X^{n-1}
 \end{aligned}
 \tag{8}$$

**2.2.1.2. Probability of a Random Variable** What is the nature of the probability of an event, or what is the relationship between probability and geometry or between the probability of an event and notions like false or true. At a first pass, various authors answer this question, one way or another. For authors like De Morgan, probability is only a degree of confidence, or credences or of belief. “By degree of probability, we really mean, or ought to mean, degree of belief” (see De Morgan, 1847, p. 172). Such a purely subjective (or personalist or Bayesian (see Bayes, 1763)) interpretation of probabilities as degrees of confidence, or credences finds its own scientific opposition, moreover, in Kolmogorov’s axiomatization of probability theory. However, perhaps we can do better, then, to think that Kolmogorov’s axiomatization of probability theory is the last word spoken on probability theory. Nobody seriously considers that Kolmogorov’s conceptual apparatus of probability theory has solved the basic problem of any probability theory, the relationship between classical logic or geometry and probability theory. One very massive disadvantage of Kolmogorov’s axiomatization of probability theory is that it is very silent especially on this issue. Any unification of geometry and probability theory into one unique mathematical framework might prove very difficult as long as we rely purely on Kolmogorov’s understanding of probability theory. It’s not surprising that the probability of an event bear at least directly, and sometimes indirectly, upon central philosophical and scientific concerns. A correct understanding of probability is one of the most important foundational scientific problems. Now let us strengthen our position with respect to the probability of an event. In our understanding, the probability of an event is something objectively and real. The probability of an event is the truth value of something or the degree to which something, i.e. a random variable X, is determined by its

own expectation value. The probability  $p(X)$  of a random variable  $X$  follows as (see equation 1)

$$\begin{aligned}
 p(X) &\equiv \frac{X \times p(X)}{X} \equiv \frac{E(X)}{X} \equiv p(X) \\
 &\equiv \frac{X \times X \times p(X)}{X \times X} \equiv \frac{X \times E(X)}{X \times X} \equiv \frac{E(X^2)}{X^2} \\
 &\equiv \frac{E(X)}{X} \equiv \frac{E(X) \times E(X)}{X \times E(X)} \equiv \frac{E(X)^2}{E(X^2)} \\
 &\equiv \frac{E(X)}{X} \equiv \frac{E(X) \times E(\underline{X})}{X \times E(\underline{X})} \equiv \frac{\sigma(X)^2}{X \times X \times (1 - p(X))} \equiv \frac{\sigma(X)^2}{E(\underline{X}^2)} \\
 &\equiv \Psi(X) \times \Psi^*(X)
 \end{aligned} \tag{9}$$

where  $\Psi(X)$  is the wave-function of  $X$ ,  $\Psi^*(X)$  is the complex conjugate wave-function of  $X$ . As soon as the probability  $p(X)$  of an event  $X$  is determined, the probability of its own other,  $1 - p(X)$ , the complementary of  $X$ , the opposite of  $X$ , anti  $X$ , is determined too. We obtain

$$\begin{aligned}
 1 - p(X) &\equiv 1 - \frac{X \times p(X)}{X} \equiv 1 - \frac{E(X)}{X} \equiv \frac{X}{X} - \frac{E(X)}{X} \equiv \frac{X - E(X)}{X} \equiv \frac{E(\underline{X})}{X} \equiv p(\underline{X}) \\
 &\equiv 1 - \frac{X \times X \times p(X)}{X \times X} \equiv 1 - \frac{X \times E(X)}{X \times X} \equiv 1 - \frac{E(X^2)}{X^2} \equiv \frac{X^2}{X^2} - \frac{E(X^2)}{X^2} \equiv \frac{X^2 - E(X^2)}{X^2} \\
 &\equiv 1 - \frac{E(X)}{X} \equiv 1 - \frac{E(X) \times E(X)}{X \times E(X)} \equiv 1 - \frac{E(X)^2}{E(X^2)} \\
 &\equiv 1 - \frac{E(X)}{X} \equiv 1 - \frac{E(X) \times E(\underline{X})}{X \times E(\underline{X})} \equiv 1 - \frac{\sigma(X)^2}{X \times X \times (1 - p(X))} \equiv 1 - \frac{\sigma(X)^2}{E(\underline{X}^2)} \\
 &\equiv 1 - \Psi(X) \times \Psi^*(X)
 \end{aligned} \tag{10}$$

### 2.2.1.3. Variance of a Random Variable

**Definition 2.4 (The Variance of a Random Variable).** *Johann Carl Friedrich Gauß (1777-1855) introduced the normal distribution and the error of mean squared in his 1809 monograph (see [Gauß, Carl Friedrich, 1809](#)). In the following, Karl Pearson (1857-1936) coined the term “standard deviation” in 1893. Pearson is writing: “Then  $\sigma$  will be termed its standard-deviation (error of mean square).” (see [Pearson, 1894](#), p. 80). Finally, the term variance was introduced by Sir Ronald Aylmer Fisher (1890-1962) in the year 1918.*

*“The ... deviations of a ... measurement from its mean ... may be ... measured by the standard deviation corresponding to the square root of the mean square error ... It is ... desirable **in analysing the causes** ... to deal with the square of the standard deviation as the measure of variability. We shall term this quantity the Variance... ”*

*(see [Fisher, Ronald Aylmer, 1919](#), p. 399)*

The deviation of a random variable  $X$  from its population mean or sample mean  $E(X)$  has a central role in statistics and is one important measure of dispersion. The variance  $\sigma(X)^2$  (see [Kolmogorov, Andreï Nikolaevich, 1950](#), p. 42), the second central moment of a distribution, is the expectation value of the squared deviation of a random variable  $X$  from its own expectation value  $E(X)$  and is determined in general as (see equation 6):

$$\begin{aligned}
 \sigma(X)^2 &\equiv E(X^2) - E(X)^2 \\
 &\equiv (X \times E(X)) - E(X)^2 \\
 &\equiv E(X) \times (X - E(X)) \\
 &\equiv E(X) \times E(\underline{X})
 \end{aligned} \tag{11}$$

while  $E(\underline{X}) \equiv X - E(X)$ . From the point of view of tensor algebra, it is

$$\begin{aligned}
 {}^2\sigma(X_{kl\mu\nu\dots}) &\equiv E({}^2X_{kl\mu\nu\dots}) - {}^2E(X_{kl\mu\nu\dots}) \\
 &\equiv (X_{kl\mu\nu\dots} \times E(X_{kl\mu\nu\dots})) - {}^2E(X_{kl\mu\nu\dots}) \\
 &\equiv E(X_{kl\mu\nu\dots}) \times (X_{kl\mu\nu\dots} - E(X_{kl\mu\nu\dots})) \\
 &\equiv E(X_{kl\mu\nu\dots}) \times E(\underline{X}_{kl\mu\nu\dots})
 \end{aligned} \tag{12}$$

while  $E(\underline{X}_{kl\mu\nu\dots}) \equiv X_{kl\mu\nu\dots} - E(X_{kl\mu\nu\dots})$ . As demonstrated by equation 12, variance depends not just on the expectation value of what has actually been observed  $E(X_{kl\mu\nu\dots})$ , but also on the expectation value that could have been observed but were not  $(E(\underline{X}_{kl\mu\nu\dots}))$ . There are circumstances in quantum mechanics where this fact is called the local hidden variable. Even if his might strike us as



peculiar, variance <sup>90</sup> is primarily a mathematical method which is of use in order to evaluate specific hypotheses in the light of some empirical facts. However, as a mathematical tool or method, variance is also a scientific description of a certain part of objective reality too. In this context, as a general mathematical principle, one fundamental meaning of variance is to provide a logically consistent link between something and its own other, between X and anti X.

“The variance in this sense is a measure of the inner contradictions of a random variable, of changes, of struggle within this random variable itself, or the greater  $\sigma(X)^2$  of a random variable, the greater the inner contradictions of this random variable. ”

(see Barukčić, 2006a, p. 57)

All things considered, we can safely say that, on the whole, **the variance is a mathematical description of the philosophical notion of the inner contradiction of a random variable X** (see Hegel, Georg Wilhelm Friedrich, 1812a, 1813, 1816) . Based on equation 11, it is

$$E(X^2) \equiv E(X)^2 + \sigma(X)^2 \quad (13)$$

or

$$\frac{E(X)^2}{E(X^2)} + \frac{\sigma(X)^2}{E(X^2)} \equiv p(X) + \frac{\sigma(X)^2}{E(X^2)} \equiv +1 \quad (14)$$

In other words, the variance (see Barukčić, 2006b) of a random variable is a determining part of the probability of a random variable. The wave function  $\Psi$  follows in general, as

$$\begin{aligned} \Psi(X) &\equiv \frac{1}{\Psi^*(X)} - \frac{\sigma(X)^2}{(\Psi^*(X) \times E(X^2))} \\ &\equiv \frac{(E(X^2) - \sigma(X)^2)}{(\Psi^*(X) \times E(X^2))} \\ &\equiv \frac{1}{(\Psi^*(X) \times E(X^2))} \times (E(X^2) - \sigma(X)^2) \\ &\equiv \frac{1}{(\Psi^*(X) \times E(X^2))} \times E(X)^2 \\ &\equiv \frac{1}{\Psi^*(X)} \times \frac{E(X)^2}{E(X^2)} \\ &\equiv \frac{1}{\Psi^*(X) \times X} \times E(X) \end{aligned} \quad (15)$$

The wave function (see Born, 1926) of a quantum-mechanical system is a central determining part of the Schrödinger wave equation (see Schrödinger, Erwin Rudolf Josef Alexander, 1926, 1929, 1952).

<sup>90</sup>Romeijn, Jan-Willem, "Philosophy of Statistics", The Stanford Encyclopedia of Philosophy (Spring 2022 Edition), Edward N. Zalta (ed.), forthcoming URL = <https://plato.stanford.edu/archives/spr2022/entries/statistics/>.

**Definition 2.5 (The First Moment Expectation of a Random Variable of  $\underline{X}$  (anti  $X$ )).** In general, let  $E(\underline{X})$  be defined as

$$E(\underline{X}) \equiv X - E(X) \equiv X - (X \times p(X)) \equiv X \times (+1 - p(X)) \quad (16)$$

and denote an expectation value of a (discrete) random variable anti  $X$  with the probability

$$p(\underline{X}) \equiv 1 - p(X) \quad (17)$$

The first moment expectation value (see [Huygens and van Schooten, 1657](#), [Kolmogorov, Andreï Nikolaevich, 1950](#), [LaPlace, 1812](#), [Whitworth, 1901](#)) of anti  $X$ , denoted as  $E(\underline{X})$ , is a number defined as follows:

$$E(\underline{X}) \equiv X - (X \times p(X)) \equiv X \times (1 - p(X)) \equiv X \times p(\underline{X}) \quad (18)$$

The first moment expectation value squared of a random variable anti  $X$  follows as

$$\begin{aligned} E(\underline{X})^2 &\equiv p(\underline{X}) \times X \times p(\underline{X}) \times X \\ &\equiv p(\underline{X}) \times p(\underline{X}) \times X \times X \\ &\equiv (p(\underline{X}) \times X)^2 \\ &\equiv E(\underline{X}) \times E(\underline{X}) \end{aligned} \quad (19)$$

**Definition 2.6 (The Second Moment Expectation of a Random Variable of  $\underline{X}$  (anti  $X$ )).** The second (see [Kolmogorov, Andreï Nikolaevich, 1950](#), p. 42) moment expectation value (or more or less arithmetic mean) of a (large) number of independent realizations of a random variable anti  $X$  follows as:

$$\begin{aligned} E(\underline{X}^2) &\equiv p(\underline{X}) \times X^2 \\ &\equiv (p(\underline{X}) \times X) \times X \\ &\equiv E(\underline{X}) \times X \\ &\equiv X \times E(\underline{X}) \end{aligned} \quad (20)$$

**Definition 2.7 (The n-th Moment Expectation of a Random Variable of  $\underline{X}$  (anti  $X$ )).** The n-th (see [Barukčić, 2020a](#), [2021c](#)) moment expectation value of a (large) number of independent realizations of a random variable anti  $X$  follows as:

$$\begin{aligned} E(\underline{X}^n) &\equiv p(\underline{X}) \times X^n \\ &\equiv (p(\underline{X}) \times X) \times X^{n-1} \\ &\equiv E(\underline{X}) \times X^{n-1} \end{aligned} \quad (21)$$

**Definition 2.8 (The Co-Variance of a Random Variable).** Sir Ronald Aylmer Fisher (1890 -1962) introduced the term covariance (see [Bailey, 1931](#)) in the year 1930 in his book as follows:

*“It is obvious too that where a considerable fraction of the variance is contributed by chance causes, the variance of any group of individuals will be inflated in comparison with the covariances between related groups ... ”*

*(see Fisher, Ronald Aylmer, 1930, p. 195)*

In general, the co-variance is defined as given by equation 22.

$$\sigma(X, Y) \equiv E(X, Y) - (E(X) \times E(Y)) \quad (22)$$

From the point of view of tensor algebra, it is

$$\sigma(X_{kl\mu\nu\dots}, Y_{kl\mu\nu\dots}) \equiv E(X_{kl\mu\nu\dots}, Y_{kl\mu\nu\dots}) - (E(X_{kl\mu\nu\dots}) \times E(Y_{kl\mu\nu\dots})) \quad (23)$$

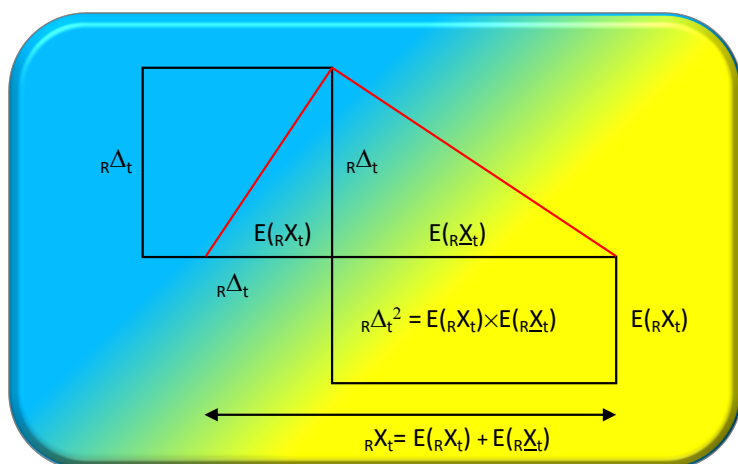
## 2.2.2. Geometry

**2.2.2.1. Euclid’s theorem** Various theories of geometry, including Euclidean geometry and non-Euclidean geometry, are based on definitions, axioms, theorems, proofs et cetera which themselves are derived more or less to some extent from knowledge of the objective reality too. Recalling Einstein’s profound position

*“... Geometrie is offenbar eine Naturwissenschaft ... Ihre Aussagen beruhen im wesentlichen auf Induktion aus der Erfahrung, nicht aber auf logischen Schlüssen. ”*

*(see Einstein, 1921, p. 6)*

and translating the same into simple English as “Geometry ... is ... a natural science ... in fact ... it ... rest essentially on induction from experience”we cannot avoid considering the limitations of geometry. In other words, explaining objective reality completely as a something like a complicated interplay between basic properties like points or lines might turn out to be stigmatized to some extent by imperfection. However, in logic, there is geometry, in geometry, there is logic. Both interpenetrate each other. The logic of geometry is determined by the geometry of logic and vice versa. Nonetheless, even if a detailed examination of geometry as presented by Euclid might reveal a number of problems, some of Euclid’s theorems are still valid. In this context, it is worth considering Euclid’s (ca. 360-280 BC) so-called right triangle theorem or Euclid’s altitude theorem or Euclid’s geometric mean theorem or simply Euclid’s theorem, published as a corollary to proposition 8 in Book VI of Euclid’s Elements (see proposition 8 in Book VI: [Euclid, of Alexandria \(300 B. C. E.\), 1908](#), p. 209) and used in proposition 14 of Book II (see Book II, proposition 14: [Euclid, of Alexandria \(300 B. C. E.\), 1908](#),



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**Figure 1. Euclid's theorem.**

Euclid's theorem

with

$${}_R X_t = E({}_R X_t) + E({}_R \underline{X}_t)$$

as the foundation of

the relationship between

${}_R X_t$  and  ${}_R \underline{X}_t$

where

$E({}_R X_t)$  is the expectation value of  ${}_R X_t$ , and

$E({}_R \underline{X}_t)$  is the expectation value of anti-  ${}_R \underline{X}_t$

pp. 409-410) to square a rectangle is defined (see Barukčić, 2013, 2015, 2016c) as

$$\begin{aligned} {}_R \Delta_t^2 &\equiv E({}_R X_t) \times E({}_R \underline{X}_t) \\ &\equiv \frac{(E({}_R X_t) \times {}_R X_t) \times (E({}_R \underline{X}_t) \times {}_R X_t)}{{}_R X_t \times {}_R X_t} \\ &\equiv \frac{({}_R a_t)^2 \times ({}_R b_t)^2}{{}_R X_t^2} \\ &\equiv \sigma(X_t)^2 \end{aligned} \quad (24)$$

where  $\sigma(X_t)^2$  is the variance of the random variable  $X_t$ . The variance  ${}_R \Delta_t^2 \equiv \sigma(X_t)^2$  of a right-angled triangle is illustrated by Fig. 1 in more detail. It is

$$\begin{aligned} {}_R \Delta_t &\equiv \frac{({}_R a_t) \times ({}_R b_t)}{{}_R X_t} \\ &\equiv \sigma(X_t) \end{aligned} \quad (25)$$

**2.2.2.1.1. Euclid's theorem and expectation value** It should be remembered, moreover, that Euclid's theorem is related to Thales of Miletus (ca. 624/623–ca.548/545 BCE) theorem. We may now apply Euclid's theorem to the relative latecomer in scientific history, the expectation values (see also fig. 1).

**Theorem 2.1** (Euclid's theorem and expectation values). *In general and according to Euclid's theorem, any random variable  ${}_R X_t$  has the potential of being in a state of superposition as*

$${}_R X_t = E({}_R X_t) + E({}_R \underline{X}_t) \quad (26)$$

where  ${}_R \Delta_t$  denotes the altitude in a right triangle and  $E({}_R X_t)$  and  $E({}_R \underline{X}_t)$  the segments on the hypotenuse  ${}_R X_t$  in a right-angle triangle. In general, something, denoted by  ${}_R X_t$ , is self-contradictory. According to Euclid's theorem, it is equally the unity and the struggle between itself  $E({}_R X_t)$  and its own other  $E({}_R \underline{X}_t)$ .

*Proof by direct proof.* The premise

$$+ 1 \equiv + 1 \quad (27)$$

is true. In the following, we rearrange the premise. We obtain

$$+ 1 + 0 \equiv + 1 + 0 \quad (28)$$

or

$$+ 1 \equiv + 1 - p(X_t) + p(X_t) \quad (29)$$

Equation 29 simplifies as

$$+ 1 \equiv p(X_t) + (+ 1 - p(X_t)) \quad (30)$$

Multiplying equation 30 by the random variable  $X_t$ , it is

$$X_t \equiv (X_t \times p(X_t)) + (X_t \times (+ 1 - p(X_t))) \quad (31)$$

Equation 31 becomes (see equation 1, p. 17)

$$X_t \equiv E(X_t) + (X_t \times (+ 1 - p(X_t))) \quad (32)$$

Equation 32 changes (see equation 16, p. 22) further. Based on Euclid's theorem, any random variable  $X_t$  is more or less in a state of superposition as given by the equation

$$X_t \equiv E(X_t) + E(\underline{X}) \quad (33)$$

□

**2.2.2.1.2. Euclid's theorem and normalisation of expectation values** The expectation values can be normalised.

**Theorem 2.2** (Euclid's theorem and normalisation of expectation values). *In general, the expectation values are normalised as*

$$+ 1 \equiv \frac{E(X_t^2)}{X_t^2} + \frac{E(\underline{X}^2)}{X_t^2} \quad (34)$$

*Proof by direct proof.* The premise

$$+ 1 \equiv + 1 \quad (35)$$

is true. In the following, we rearrange the premise. We obtain

$$X_t \equiv X_t \quad (36)$$

Equation 36 changes (see equation 33, p. 25) slightly. It is

$$X_t \equiv E(X_t) + E(\underline{X}) \quad (37)$$

Multiplying equation 37 by  $X_t$ , it is

$$X_t \times X_t \equiv (X_t \times E(X_t)) + (X_t \times E(\underline{X})) \quad (38)$$

or (see equation 6, p. 17) and equation 20, p. 22)

$$X_t^2 \equiv E(X_t^2) + E(\underline{X}^2) \quad (39)$$

Normalising the relationships of equation 39, it is

$$+1 \equiv \frac{X_t^2}{X_t^2} \equiv \frac{E(X_t^2)}{X_t^2} + \frac{E(\underline{X}^2)}{X_t^2} \quad (40)$$

□

### 2.2.2.1.3. Euclid's theorem and normalisation of probabilities

**Theorem 2.3.** *Euclid's theorem can be normalized. In general, it is*

$$p({}_R X_t) + p({}_R \underline{X}_t) = +1 \quad (41)$$

*Proof.* **If** the premise

$$+1 = +1 \quad (42)$$

is true, **then** the conclusion

$$p({}_R X_t) + p({}_R \underline{X}_t) = +1 \quad (43)$$

is also true, the absence of any technical errors and other errors of human reasoning presupposed. The starting point of this proof (premise:  $+1 = +1$ ) is true. Multiplying Eq. 42 by  ${}_R X_t$  it is

$$+{}_R X_t = +{}_R X_t \quad (44)$$

Rearranging Eq. 44, we obtain

$$+{}_R X_t - E({}_R X_t) + E({}_R X_t) = +{}_R X_t + 0 \quad (45)$$

while it is necessary that  $E({}_R X_t)$  is for sure one determining part of  ${}_R X_t$ , whatever  $E({}_R X_t)$  and  ${}_R X_t$  may denote. In general, we consider without an exception all but  $E({}_R X_t)$  at a certain period of or point in time  $t$  as anti  $E({}_R X_t)$ . Anti  $E({}_R X_t)$  is denoted by  $E({}_R \underline{X}_t)$ . Arithmetically, we define  $E({}_R \underline{X}_t)$  as

$$E({}_R \underline{X}_t) \equiv +({}_R X_t) - E({}_R X_t) \quad (46)$$

Eq. 45 changes in perfect agreement with 46 to

$$+E({}_R \underline{X}_t) + E({}_R X_t) = {}_R X_t \quad (47)$$

By rearranging Eq. 47, we obtain the general normalized form of Euclid's theorem as

$$+ \left( \frac{E({}_R X_t)}{{}_R X_t} \right) + \left( \frac{E(\underline{{}_R X_t})}{\underline{{}_R X_t}} \right) = \left( \frac{{}_R X_t}{{}_R X_t} \right) = +1 \quad (48)$$

From the point of view of geometry, the probability of a single event, an entity, a quantity, a number et cetera is the extent to which  $E({}_R X_t)$ , this single event, entity, quantity, number et cetera, is a determining part of  ${}_R X_t$ . In general, it is

$$p({}_R X_t) \equiv \frac{E({}_R X_t)}{{}_R X_t} \quad (49)$$

From the point of view of geometry, the probability of a single anti-event, an anti-entity, an anti-quantity, an anti-number et cetera is the extent to which  $E(\underline{{}_R X_t})$ , this single anti-event, an anti-entity, an anti-quantity, an anti-number et cetera, is a determining part of  $\underline{{}_R X_t}$ . In general, it is

$$p(\underline{{}_R X_t}) \equiv \frac{E(\underline{{}_R X_t})}{\underline{{}_R X_t}} = 1 - \frac{({}_R X_t) \times p({}_R X_t)}{{}_R X_t} = 1 - \frac{E({}_R X_t)}{{}_R X_t} = 1 - p({}_R X_t) \quad (50)$$

Taking into account the previous definitions (Eq. 49 and Eq. 50) then Eq. 48 changes to

$$p({}_R X_t) + p(\underline{{}_R X_t}) = +1 \quad (51)$$

□

#### **Theorem 2.4 (THE APPROXIMATE PROBABILITY OF AN EVENT).**

In general, it is

$$p({}_R X_t) \equiv \exp^{-p(\underline{{}_R X_t})} \quad (52)$$

*Proof.* **If** the premise

$$+1 = +1 \quad (53)$$

is true, **then** the conclusion

$$p({}_R X_t) \equiv \exp^{-p(\underline{{}_R X_t})} \quad (54)$$

is also true, the absence of any technical errors and other errors of human reasoning presupposed. The starting point of this proof (premise:  $+1 = +1$ ) is true. Multiplying Eq. 53 by the probability  $p({}_R X_t)$  of an event  ${}_R X_t$  at the (period of) time / Bernoulli trial  $t$ , it is

$$p({}_R X_t) \equiv p({}_R X_t) \quad (55)$$

Eq. 55 changes according to Eq. 51 into

$$p({}_R X_t) \equiv (+1 - p(\underline{{}_R X_t})) \quad (56)$$

or to

$$p({}_R X_t) \equiv \left( +1 - \left( \frac{n \times p({}_R X_t)}{n} \right) \right) \quad (57)$$

Assumed that the probability is constant from trial to trial while the number of observations increases, we obtain the following.

$$p({}_R X_t)^n \equiv \left( +1 - \left( \frac{n \times p({}_R X_t)}{n} \right) \right)^n \quad (58)$$

Eq. 58 can be simplified as

$$p({}_R X_t)^n \equiv \left( +1 - \left( \frac{E({}_R X_t)}{n} \right) \right)^n \quad (59)$$

From elementary calculus (see also DeGroot and Schervish, 2005, p. 195) it is known that

$$\lim_{n \rightarrow +\infty} \left( +1 - \left( \frac{E({}_R X_t)}{n} \right) \right)^n \equiv \exp^{-E({}_R X_t)} \quad (60)$$

According to Eq. 60, Eq. 59 is rearranged as

$$p({}_R X_t)^n \equiv \exp^{-E({}_R X_t)} \quad (61)$$

The probability of a single event follows as

$$\begin{aligned} p({}_R X_t) &\equiv \sqrt[n]{p({}_R X_t)^n} \\ &\equiv \sqrt[n]{\exp^{-E({}_R X_t)}} \\ &\equiv \exp^{\frac{-E({}_R X_t)}{n}} \\ &\equiv \exp^{-\frac{(n \times p({}_R X_t))}{n}} \end{aligned} \quad (62)$$

Finally, the probability of a single event (see Barukčić, 2019e, pp. 1843-1844) is given by

$$p({}_R X_t) \equiv \exp^{-p({}_R X_t)} \quad (63)$$

□

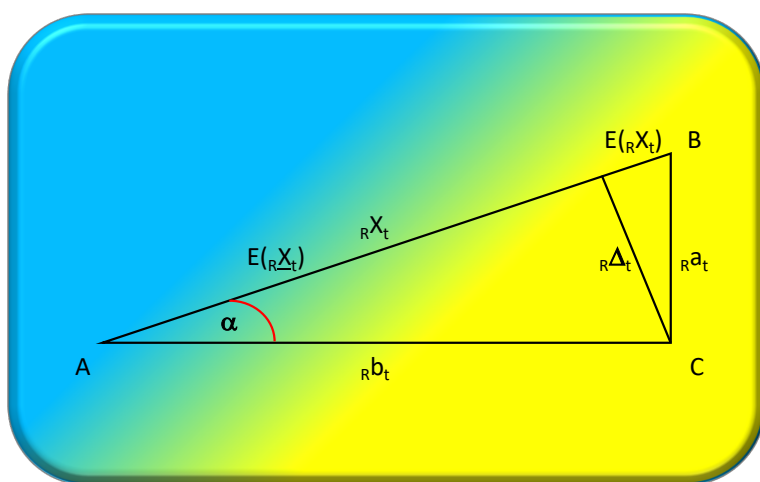
Our sun has risen every day for a long time in the past. However, will the same sun rise tomorrow, for sure? In the light of such empirical facts, any inference from the known or observed to the unknown or unobserved, has become known as “inductive inferences”. Inductive inference is often overshadowed by the possibility of being mistaken and is associated with a certain level of significance (see Arbuthnot, John, 1710, Venn, 1888), often denoted as the p-value (see Pearson, 1900b). Historically, it was especially David Hume<sup>91</sup> who put into question in his 1739 Book ‘A Treatise of Human Nature, part iii, section 6’ (see Hume, 1739) any justification in which humans form knowledge which became known as Hume’s ‘problem of induction’.

<sup>91</sup>Henderson, Leah, “The Problem of Induction”, The Stanford Encyclopedia of Philosophy (Spring 2020 Edition), Edward N. Zalta (ed.), URL = <https://plato.stanford.edu/archives/spr2020/entries/induction-problem/>.



**2.2.2.2. Pythagorean theorem** Pythagoras of Samos (c. 570 – c. 495 BCE) lived on the island of Samos in the Aegean Sea, in Egypt, Babylon and southern Italy. In mathematics, Pythagoras is credited with several scientific and mathematical discoveries, including the Pythagorean theorem, or Pythagoras' theorem, too. However, the history of Pythagorean theorem is more or less the subject of much debate while neither the date of first discovery of Pythagoras' theorem nor the date of the first proof of Pythagoras' theorem is certain. At present, there are quite a few publications available suggesting that the Pythagorean theorem was known in ancient Babylon, Egypt and India (the Baudhayana Shulba Sutra), too. Yet astonishingly enough, there are reports that the Pythagorean theorem was found on an old Babylonian tablet meanwhile known as Plimpton 322 (see Friberg, 1981, Maor, 2007), written between 1790 and 1750 BCE during the reign of King Hammurabi the Great.

**Definition 2.9 (The right-angled triangle).** A right-angled triangle is a triangle in which one angle is a 90-degree angle. Let  ${}_R X_t$  denote the hypotenuse, the side opposite the right angle (side  ${}_R X_t$  inside figure 2). The sides  ${}_R a_t$  and  ${}_R b_t$  are called legs of the triangle. In a right-angled triangle ABC, the side AC, which is abbreviated as  ${}_R b_t$ , is the side which is adjacent to the angle  $\alpha$ , while the side CB, denoted as  ${}_R a_t$ , is the side opposite to the angle  $\alpha$ . Figure 2 might illustrate a right-angled triangle (see Bettinger and Englund, 1960). The relation between the sides and angles of a right-angled triangle are known to be the basis for trigonometry, but are the basis of probability theory too.



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**Figure 2. Right-angled triangle.**

**Right-angled triangle.**

where

${}_R X_t$  is hypotenuse, and

${}_R a_t$  and  ${}_R b_t$

are called triangles legs.

Again,  ${}_R X_t$  is in the state of superposition, a law which has been re-formulated by the Danish geologist Nicolaus Steno (see Stenonis, Nicolai, 1669) in his 1696 book 'De Solido Intra Naturaliter Contento Dissertationis Prodomus'. Thus far, how big is the chance or probability that three random points like A, B, C in space-time are able to form a certain, stable right-angled triangle? Problems of similar type have been studied in the 18th century under the notion of geometric probability (see Milman, Vitali D., 2008, Solomon, 1978). Geometry and probability are deeply interrelated. No wonder, there is something extremely simple and deeply hidden even inside Einstein's masterpiece, the tremendously complex general theory of relativity. That turns out to be the right-angled triangle (ninety-degree angle at one of its corner).

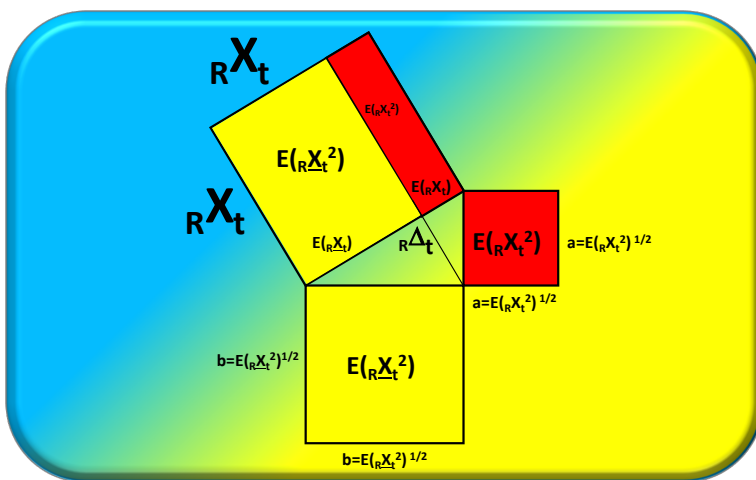
2.2.2.2.1. Pythagorean theorem in general

Definition 2.10 (The Pythagorean theorem).

The famous Pythagorean theorem of Euclidean geometry is attributed to the Greek thinker Pythagoras of Samos (ca. 570 – ca. 495 BCE). However, even if attributed to Pythagoras, the theorem has been known to the Babylonians (see Maor, 2007) more than a thousand years before Pythagoras. In general, the Pythagorean theorem is defined as

$${}_R a_t^2 + {}_R b_t^2 \equiv {}_R X_t^2 \tag{64}$$

where  ${}_0$  may denote the point of view of a co-moving observer, while  ${}_R$  may denote the point of view of a stationary observer at a certain point in space-time  $t$ . Fig. 3 is illustrating the Pythagorean theorem in all its splendour and beauty in more detail.



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Figure 3. The Pythagorean theorem and statistics/probability theory.

Pythagorean theorem and probability theory. In general, it is

$$({}_R X_t)^2 = E({}_R X_t^2) + E({}_R X_t^2)$$

2.2.2.2.2. Pythagorean theorem normalised

Theorem 2.5. The normalised Pythagorean theorem is determined as

$$\frac{{}_R a_t^2}{{}_R X_t^2} + \frac{{}_R b_t^2}{{}_R X_t^2} \equiv +1^{+2} \tag{65}$$

Proof. If the premise

$$+ 1 \equiv +1 \tag{66}$$

is true, then the conclusion

$$\frac{{}_R a_t^2}{{}_R X_t^2} + \frac{{}_R b_t^2}{{}_R X_t^2} \equiv +1^{+2} \tag{67}$$

is also true, the absence of any technical errors and other errors of human reasoning presupposed. The starting point of this proof (premise:  $+1 = +1$ ) is true. The Pythagorean theorem is proofed (see Eq. 64) as  ${}_R a_t^2 + {}_R b_t^2 \equiv {}_R X_t^2$ . Eq. 66 changes to

$${}_R X_t^2 \equiv {}_R X_t^2 \quad (68)$$

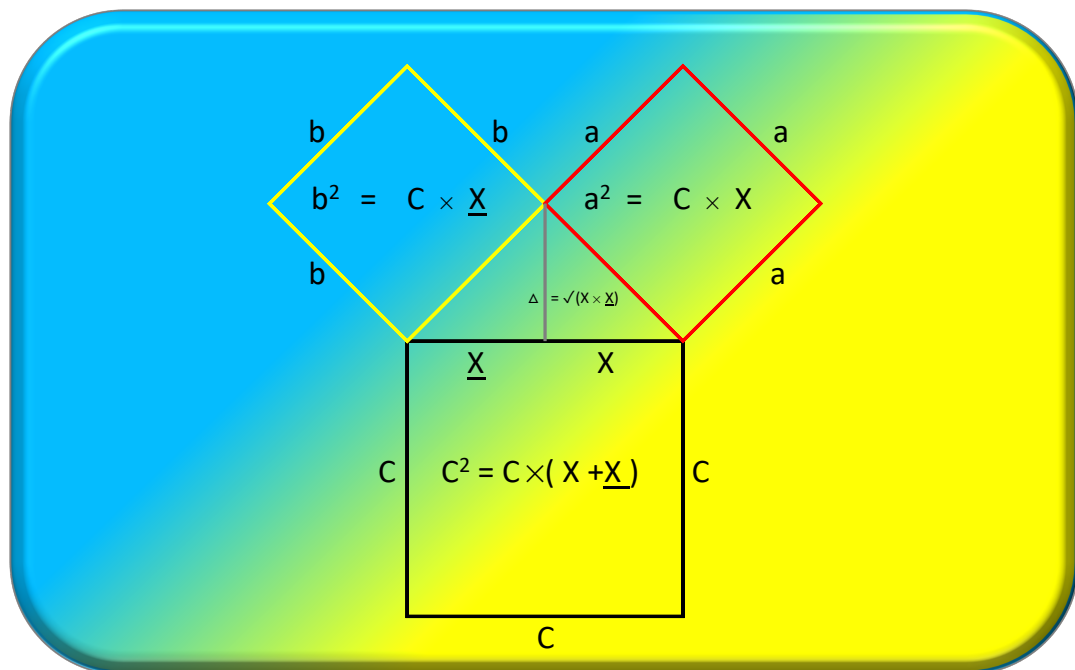
and finally to

$${}_R a_t^2 + {}_R b_t^2 \equiv {}_R X_t^2 \equiv C^2 \quad (69)$$

In the following, We set  $C = {}_R X_t$  and  $a = {}_R a_t$  and  $b = {}_R b_t$ . Dividing Eq. 69 by  ${}_R X_t^2$  under conditions where this is possible and allowed, we obtain the normalized form of the Pythagorean theorem (see equation 4, p. 31) as

$$\frac{{}_R a_t^2}{{}_R X_t^2} + \frac{{}_R b_t^2}{{}_R X_t^2} \equiv \frac{{}_R X_t^2}{{}_R X_t^2} + 1^{+2} \quad (70)$$

□



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**Figure 4. Geometry and probability theory.**

In our understanding, there are conditions where probability theory / statistics is related with geometry

(i.e. Pythagorean theorem, Euclid's theorem et cetera) (see also figure 4) by the equation:

$$a^2 \equiv E(X^2) \quad (71)$$

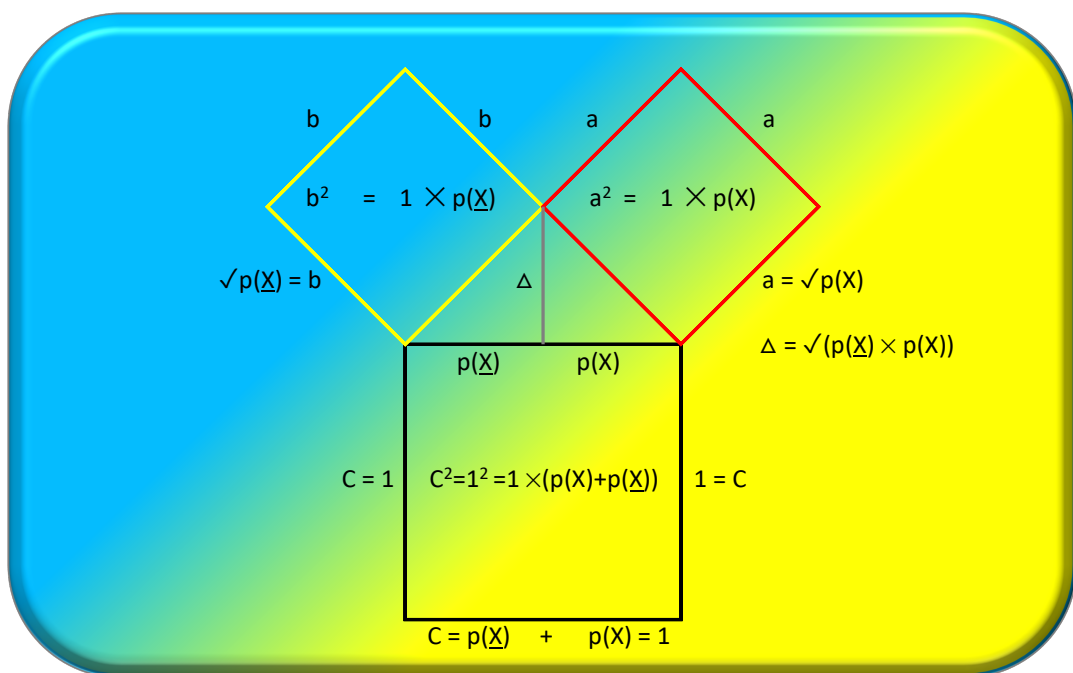
Further research should be able and might provide convincing evidence whether - and to what extent - equation 71 makes any sense at all. However, none of these reliefs us of our duty to seriously consider the possibility of negative probabilities (see theorem 3.38 Barukčić, 2019b, pp. 67-68) like

$$-p(X) \equiv \frac{-E(X)}{-X} \quad (72)$$

It is

$$+1 \equiv p(X) + 1 - p(X) \equiv p(X) + p(\underline{X}) \equiv C \quad (73)$$

as illustrated by figure 5



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**Figure 5. Geometry and probability theory.**

and equally

$$\begin{aligned} +1^{+2} &\equiv (1 \times p(X)) + (1 \times (1 - p(X))) \equiv (1 \times (p(X) + p(\underline{X}))) \equiv C^2 \\ &\equiv a^2 + b^2 \equiv C^2 \\ &\equiv C^2 \end{aligned} \quad (74)$$

**2.2.2.2.3. Geometry and probability** The distributions of properties of geometric objects like length, area, volume, etc. is studied by **geometric probability** (see [Klain and Rota, 1997](#), [Milman, Vitali D., 2008](#), [Solomon, 1978](#)) too. In other words, probability is involved in geometry. **Example.** Let the length of a line  $C$  be  $C = 10$  cm. Let  $X$  denote the length of a sub-line of  $C$ . Let  $X = 5$  cm. The probability  $p(C = X) = 5 / 10 = 1/2$ . However, as can be seen by figure 4, probability and geometry are not only deeply interrelated. In contrast to Menger's approach to **probabilistic geometry** (see [Menger, 1951, 2003](#), [Milman, Vitali D., 2008](#), [Špaček, 1956](#)), probability theory can be defined by geometry, completely and potentially vice versa too. The trigonometrical functions are the geometric way to formulate probability.

In consideration of the preceding of figure 5 before and the general definition of the trigonometric function sine ( see also Abu Dscha'far Muhammad ibn Musa al Chwarizmi's Algebra, written around 825 CE translated by Gerard of Cremona (1114-1187) from Arabic into Latin ), denoted as  $\sin$ , it is

$$\sin \alpha \equiv \frac{a}{c} \equiv \frac{a}{1} \equiv \frac{\sqrt[2]{p(X)}}{1} \equiv \sqrt[2]{p(X)} \quad (75)$$

and

$$\sin^2 \alpha \equiv (\sin \alpha)^2 \equiv \sin \alpha \times \sin \alpha \equiv \left(\frac{a}{c}\right)^2 \equiv \left(\frac{a}{1}\right)^2 \equiv a^2 \equiv p(X) \equiv \Psi(X) \times \Psi^*(X) \quad (76)$$

Against the background of figure 5 and the general definition of the function cosecant, denoted as  $\csc$ , it is

$$\csc \alpha \equiv \frac{c}{a} \equiv \frac{1}{a} \equiv \frac{1}{\sqrt[2]{p(X)}} \quad (77)$$

and equally

$$\csc^2 \alpha \equiv (\csc \alpha)^2 \equiv \csc \alpha \times \csc \alpha \equiv \left(\frac{c}{a}\right)^2 \equiv \left(\frac{1}{a}\right)^2 \equiv \frac{1}{a^2} \equiv \frac{1}{p(X)} \quad (78)$$

In general it is

$$\sin \alpha \times \csc \alpha \equiv +1 \quad (79)$$

In the light of figure 5 above, and the definition the function cosine, denoted as  $\cos$ , it is

$$\cos \alpha \equiv \frac{b}{c} \equiv \frac{b}{1} \equiv \frac{\sqrt[2]{p(X)}}{1} \equiv \sqrt[2]{p(X)} \quad (80)$$

and at the same time

$$\cos^2 \alpha \equiv (\cos \alpha)^2 \equiv \cos \alpha \times \cos \alpha \equiv \left(\frac{b}{c}\right)^2 \equiv \left(\frac{b}{1}\right)^2 \equiv b^2 \equiv p(X) \equiv 1 - \Psi(X) \times \Psi^*(X) \quad (81)$$

Claudius Ptolemy (c. 85 – c. 165 CE) was the most influential Greek astronomers of his time, Ptolemy developed a geocentric theory of our solar system that prevailed for more than 1400 years

until overthrown by Isaac Newton's (see [Newton, 1687](#)) worldview. Already Ptolemy knew about the relationship

$$\sin^2 \alpha + \cos^2 \alpha \equiv +1 \quad (82)$$

which is meanwhile identified in more detail as

$$\sin^2 \alpha + \cos^2 \alpha \equiv p(X) + p(\underline{X}) \equiv +1 \quad (83)$$

We are justified in asking whether the expectation value of an angle  $\alpha$ , denoted as  $E(\alpha)$ , might be given by the equation

$$E(\alpha) \equiv \alpha \times (\sin^2 \alpha) \quad (84)$$

whether  $E(\alpha^2)$  would be given by the equation

$$E(\alpha^2) \equiv \alpha \times \alpha \times (\sin^2 \alpha) \quad (85)$$

Under these assumptions, the variance  $\sigma(\alpha)^2$  of an angle would follow as

$$\sigma(\alpha)^2 \equiv E(\alpha^2) - E(\alpha)^2 \equiv \alpha \times \alpha \times (\sin^2 \alpha) \times (1 - (\sin^2 \alpha)) \quad (86)$$

Having regard to figure 5 above and on the basis of the definition of the function secant, denoted by sec, it is

$$\sec \alpha \equiv \frac{c}{b} \equiv \frac{1}{\frac{b}{c}} \equiv \frac{1}{\sqrt[2]{p(\underline{X})}} \quad (87)$$

and equally

$$\sec^2 \alpha \equiv (\sec \alpha)^2 \equiv \sec \alpha \times \sec \alpha \equiv \left(\frac{c}{b}\right)^2 \equiv \left(\frac{1}{\frac{b}{c}}\right)^2 \equiv \frac{1}{p(\underline{X})} \quad (88)$$

$$\cos \alpha \times \sec \alpha \equiv +1 \quad (89)$$

On the basis of a presentation by figure 5 and the known definition of the function tangent, denoted as tan, it is

$$\tan \alpha \equiv \frac{\sin \alpha}{\cos \alpha} \equiv \frac{\frac{a}{c}}{\frac{b}{c}} \equiv \frac{a}{b} \equiv \frac{\sqrt[2]{p(\underline{X})}}{\sqrt[2]{p(\underline{X})}} \equiv \sqrt[2]{\frac{p(\underline{X})}{p(\underline{X})}} \quad (90)$$

and equally

$$\tan^2 \alpha \equiv (\tan \alpha)^2 \equiv (\tan \alpha) \times (\tan \alpha) \equiv \left(\frac{\sin \alpha}{\cos \alpha}\right) \times \left(\frac{\sin \alpha}{\cos \alpha}\right) \equiv \frac{\sin^2 \alpha}{\cos^2 \alpha} \equiv \frac{\frac{a^2}{c^2}}{\frac{b^2}{c^2}} \equiv \frac{a^2}{b^2} \equiv \frac{p(\underline{X})}{p(\underline{X})} \quad (91)$$

In view of figure 5 and the definition of cotangent, denoted as  $\cot$ , it is

$$\cot \alpha \equiv \frac{b}{a} \equiv \frac{\sqrt[2]{p(X)}}{\sqrt[2]{p(X)}} \equiv \sqrt[2]{\frac{p(X)}{p(X)}} \quad (92)$$

Furthermore, it is

$$\cot^2 \alpha \equiv (\cot \alpha)^2 \equiv (\cot \alpha) \times (\cot \alpha) \equiv \cot^2 \alpha \equiv \frac{b^2}{a^2} \equiv \frac{p(X)}{p(X)} \quad (93)$$

Based on the findings as explained before and by figure 5 it is

$$\tan \alpha \times \cot \alpha \equiv +1 \quad (94)$$

An undeniable consequence of the previous explanations is that the “local hidden variable” (see Bohm, 1952, De Broglie, Louis, 1927), denoted as  $E(X)$ , is determined by the relationship

$$E(X) \equiv X \times \cos^2 \alpha \equiv \frac{\sigma(X)^2}{E(X)} \equiv \frac{\sigma(X)^2}{\Psi(X) \times X \times \Psi^*(X)} \quad (95)$$

while the variance from the point of view of geometry is given as

$$\begin{aligned} \sigma(X)^2 &\equiv E(X^2) - E(X)^2 \\ &\equiv (X \times p(X)) \times X \times (1 - p(X)) \\ &\equiv (X \times \sin^2 \alpha) \times X \times (1 - \sin^2 \alpha) \\ &\equiv (X \times \sin^2 \alpha) \times X \times (\cos^2 \alpha) \\ &\equiv X^2 \times (\sin^2 \alpha) \times (\cos^2 \alpha) \end{aligned} \quad (96)$$

From the point of view of tensor algebra, we obtain

$$\begin{aligned} p(X_{kl\mu\nu\dots}) &\equiv \frac{X_{kl\mu\nu\dots} \times p(X_{kl\mu\nu\dots})}{X_{kl\mu\nu\dots}} \equiv \frac{E(X_{kl\mu\nu\dots})}{X_{kl\mu\nu\dots}} \\ &\equiv \frac{X_{kl\mu\nu\dots} \times X_{kl\mu\nu\dots} \times p(X_{kl\mu\nu\dots})}{X_{kl\mu\nu\dots} \times X_{kl\mu\nu\dots}} \equiv \frac{E(^2X_{kl\mu\nu\dots})}{^2X_{kl\mu\nu\dots}} \\ &\equiv \frac{E(X_{kl\mu\nu\dots}) \times E(X_{kl\mu\nu\dots})}{E(X_{kl\mu\nu\dots}) \times X_{kl\mu\nu\dots}} \equiv \frac{^2E(X_{kl\mu\nu\dots})}{E(^2X_{kl\mu\nu\dots})} \\ &\equiv \Psi(X_{kl\mu\nu\dots}) \times \Psi^*(X_{kl\mu\nu\dots}) \end{aligned} \quad (97)$$

where  $\Psi(X_{kl\mu\nu\dots})$  is the wave-function tensor of  $X_{kl\mu\nu\dots}$ ,  $\Psi^*(X_{kl\mu\nu\dots})$  is the complex conjugate wave-function tensor of  $X_{kl\mu\nu\dots}$ .

#### 2.2.2.2.4. Pythagorean theorem and negation

**Theorem 2.6.** In general,  ${}_R a_t$  is the negation of  ${}_R b_t$  and vice versa. It is

$${}_R a_t^2 = \neg({}_R b_t) \times {}_R X_t^2 \quad (98)$$

*Proof.* **If** the premise

$$+1 = +1 \quad (99)$$

is true, **then** the conclusion

$${}_R a_t = \sqrt[2]{\left(+1^{+2} - \frac{{}_R b_t^2}{{}_R X_t^2}\right)} \times {}_R X_t \quad (100)$$

is also true, the absence of any technical errors and other errors of human reasoning presupposed. The starting point of this proof (premise:  $+1 = +1$ ) is true. Eq. 99 is rearranged as

$$+1^{+2} = +1^{+2} \quad (101)$$

The normalized form of the Pythagorean theorem is proofed as (see theorem 2.5, Eq. 70) as  $\frac{{}_R a_t^2}{{}_R X_t^2} +$

$\frac{{}_R b_t^2}{{}_R X_t^2} = +1^{+2}$ . Eq. 101 changes to

$$\frac{{}_R a_t^2}{{}_R X_t^2} + \frac{{}_R b_t^2}{{}_R X_t^2} = +1^{+2} \quad (102)$$

Rearranging Eq. 102

$$\frac{{}_R a_t^2}{{}_R X_t^2} + = \left(+1^{+2} - \frac{{}_R b_t^2}{{}_R X_t^2}\right) \quad (103)$$

Simplifying Eq. 103, it is

$${}_R a_t^2 \times +1^{+2} = \left(+1^{+2} - \frac{{}_R b_t^2}{{}_R X_t^2}\right) \times {}_R X_t^2 \quad (104)$$

Eq. 104 changes to

$${}_R a_t^2 = \left(+1^{+2} - \frac{{}_R b_t^2}{{}_R X_t^2}\right) \times {}_R X_t^2 \quad (105)$$

and to

$${}_R a_t = \sqrt[2]{\left(+1^{+2} - \frac{{}_R b_t^2}{{}_R X_t^2}\right)} \times {}_R X_t \quad (106)$$

We define in general

$$\neg({}_R b_t) \equiv \left(+1^{+2} - \frac{{}_R b_t^2}{{}_R X_t^2}\right) \quad (107)$$

Eq. 105 changes to

$${}_R a_t^2 = \neg({}_R b_t) \times {}_R X_t^2 \quad (108)$$

□



The negation of  ${}_R b_t$  need to be calculated similarly. We will obtain

$$\neg({}_R a_t) \equiv \left( +1^{+2} - \frac{{}_R a_t^2}{{}_R X_t^2} \right) \quad (109)$$

Under conditions of Einstein's special relativity where  ${}_R a_t$  does denote the rest-mass and where  ${}_R X_t$  does denote the relativistic mass, we obtain the identity with reciprocal Lorentz factor or Lorentz term (see also Lorentz, 1899, p. 432) as  $\left( \sqrt[2]{\left( +1^{+2} - \frac{{}_R b_t^2}{{}_R X_t^2} \right)} \right) \equiv \left( \sqrt[2]{\left( 1 - \frac{v^2}{c^2} \right)} \right)$  (see also Barukčić, 2019a).

**2.2.2.2.5. The n-dimensional Pythagorean theorem** The n-dimensional Pythagorean theorem can be derived in a simple and logically consistent way.

**Theorem 2.7.** *The n-dimensional Pythagorean theorem is determined as*

$${}_R a_t^{2n} + {}_R b_t^{2n} \equiv {}_R X_t^{2n} \quad (110)$$

*Proof.* **If** the premise

$$+1 = +1 \quad (111)$$

is true, **then** the conclusion

$${}_R a_t^{2n} + {}_R b_t^{2n} \equiv {}_R X_t^{2n} \quad (112)$$

is also true, the absence of any technical errors and other errors of human reasoning presupposed. The starting point of this proof (premise:  $+1 = +1$ ) is true. Eq. 111 change quickly to

$${}_R X_t^2 = {}_R X_t^2 \quad (113)$$

Multiplying Eq. 113 by Eq. 65 of theorem 2.5 known to be derived as  $\left( \frac{{}_R a_t^2}{{}_R X_t^2} + \frac{{}_R b_t^2}{{}_R X_t^2} = {}_R X_t^2 \right)$  yields

$$\frac{{}_R a_t^2}{{}_R X_t^2} + \frac{{}_R b_t^2}{{}_R X_t^2} = {}_R X_t^2 \quad (114)$$

Several properties of the Pythagorean theorem are already identified. In general, it is proofed that

$${}_R a_t^2 \equiv E({}_R X_t) \times {}_R X_t \quad (115)$$

or that

$${}_R a_t^{2n} \equiv E({}_R X_t)^n \times {}_R X_t^n \equiv (E({}_R X_t) \times {}_R X_t)^n \quad (116)$$

Furthermore, it is

$${}_R b_t^2 \equiv E({}_R X_t) \times {}_R X_t \quad (117)$$

and equally

$${}_R b_t^{2n} \equiv E({}_R X_t)^n \times {}_R X_t^n \equiv (E({}_R X_t) \times {}_R X_t)^n \quad (118)$$

where  $n$  might denote the number of dimensions. Rearranging Eq. 114 according to the relationship of Eq. 115 it is

$$\frac{E({}_R X_t) \times {}_R X_t}{{}_R X_t^2} + \frac{{}_R b_t^2}{{}_R X_t^2} = +1^2 \quad (119)$$

Rearranging Eq. 119 according to the relationship of Eq. 117 it is

$$\frac{E({}_R X_t) \times {}_R X_t}{{}_R X_t^2} + \frac{E({}_R \underline{X}_t) \times {}_R X_t}{{}_R X_t^2} = +1^2 \quad (120)$$

Eq. 120 simplifies further as

$$\frac{E({}_R X_t)}{{}_R X_t} + \frac{E({}_R \underline{X}_t)}{{}_R X_t} = +1^2 \quad (121)$$

Simplifying Eq. 121 it is

$$E({}_R X_t) + E({}_R \underline{X}_t) = {}_R X_t^1 \times 1^1 \times 1^1 = ({}_R X_t \times 1 \times 1)^1 = {}_R X_t^1 = {}_R X_t \quad (122)$$

As known, it is ( $U^1 \times U^0 \equiv U^{+1} \equiv U$ ). However, Eq. 122 simplifies further. The most simple and most general form of the Pythagorean theorem (see Barukčić, 2016c) is based on the fundamental relationship,

$$E({}_R X_t) + E({}_R \underline{X}_t) \equiv {}_R X_t \quad (123)$$

In particular, the Pythagorean theorem can be extended to higher dimensions (see Yeng et al., 2008) too. In the  $n$ -dimensional case (see Barukčić, 2020b), the relationship before becomes

$$(E({}_R X_t) + E({}_R \underline{X}_t))^n \equiv {}_R X_t^n \quad (124)$$

Multiplying Eq. 124 by  ${}_R X_t^n$ , the Pythagorean theorem becomes something like

$$(E({}_R X_t) + E({}_R \underline{X}_t))^n \times {}_R X_t^n \equiv {}_R X_t^n \times {}_R X_t^n \quad (125)$$

or as

$$\underbrace{E({}_R X_t)^n \times {}_R X_t^n}_{{}_R a_t^{2n}} + \underbrace{\dots}_{{}_R b_t^{2n}} \equiv {}_R X_t^n \times {}_R X_t^n \equiv {}_R X_t^{2n} \quad (126)$$

In general, the  $n$ -dimensional Pythagorean theorem is determined as

$${}_R a_t^{2n} + {}_R b_t^{2n} \equiv {}_R X_t^{2n} \quad (127)$$

□

### 2.2.2.2.6. Pythagorean theorem and probability of an event

#### Theorem 2.8 (PYTHAGOREAN THEOREM AND PROBABILITY OF AN EVENT).

Under conditions of special theory of relativity (see also [Einstein, 1905](#)), the probability that  ${}_R E_t$  is determined by  ${}_0 E_t$  is given by

$$p({}_R E_t) \equiv \left( +1 - \left( \frac{v^2}{c^2} \right) \right) \quad (128)$$

by direct proof. According to Eq. 115 on page 37 it is

$${}_R a_t^2 \equiv E({}_R X_t) \times {}_R X_t \quad (129)$$

Eq. 129 is equivalent with

$${}_R a_t^2 \equiv p({}_R X_t) \times {}_R X_t \times {}_R X_t \quad (130)$$

Dividing Eq. 130 by  ${}_R X_t^2$  it is

$$\frac{{}_R a_t^2}{({}_R X_t \times {}_R X_t)} \equiv \frac{p({}_R X_t) \times ({}_R X_t \times {}_R X_t)}{({}_R X_t \times {}_R X_t)} \equiv p({}_R X_t) \quad (131)$$

Let us consider conditions of the special theory of relativity where  ${}_R a_t^2 \equiv {}_0 E_t^2 \equiv {}_R E_t^2 \times \left( +1 - \left( \frac{v^2}{c^2} \right) \right)$ . Furthermore, there are conditions where  ${}_R X_t \equiv {}_R E_t$  and it follows that Eq. 131 changes to

$$\begin{aligned} p({}_R E_t) &\equiv \frac{{}_R a_t^2}{({}_R X_t \times {}_R X_t)} \\ &\equiv \frac{{}_0 E_t^2}{({}_R E_t^2)} \equiv \frac{{}_R E_t^2 \times \left( +1 - \left( \frac{v^2}{c^2} \right) \right)}{({}_R E_t^2)} \\ &\equiv \left( +1 - \left( \frac{v^2}{c^2} \right) \right) \end{aligned} \quad (132)$$

Under conditions of special theory of relativity, the probability that total energy (relativistic energy et cetera)  ${}_R E_t$  is determined by the rest-energy  ${}_0 E_t$  is given by

$$p({}_R E_t = {}_0 E_t) \equiv \left( +1 - \left( \frac{v^2}{c^2} \right) \right) \quad (133)$$

□

**Remark 2.1.** *It need not be noisy to consider whether there exist any circumstances which might permit us to conclude that  $p({}_R E_t = {}_0 E_t) \equiv \left( +1 - \left( \frac{v^2}{c^2} \right) \right)$  indicates the probability to which a quantum mechanical entity can be regarded as being local.*

**2.2.2.2.7. Pythagorean theorem and the wave function  $\Psi$**  Especially in order to compute how a wave propagates and behaves like in quantum mechanics, the application of the superposition principle is of advantage. There is some evidence that **the superposition principle** has been stated by Daniel Bernoulli (1700 – 1782) in 1753 (“**Later (1753), Daniel Bernoulli formulated the principle of superposition ...**” (see [Leon Brillouin, 1946](#), p. 2)).

**Theorem 2.9.** *In general, it is*

$$\left( \frac{\Psi({}_R X_t) \times \Psi(E({}_R X_t))}{\Psi({}_R X_t) \times \Psi({}_R X_t)} \right) + \left( \frac{\Psi({}_R X_t) \times \Psi(E({}_R \underline{X}_t))}{\Psi({}_R X_t) \times \Psi({}_R X_t)} \right) \equiv +1^2 \quad (134)$$

*Proof.* **If** the premise

$$+1 = +1 \quad (135)$$

is true, **then** the conclusion

$$\left( \frac{\Psi({}_R X_t) \times \Psi(E({}_R X_t))}{\Psi({}_R X_t) \times \Psi({}_R X_t)} \right) + \left( \frac{\Psi({}_R X_t) \times \Psi(E({}_R \underline{X}_t))}{\Psi({}_R X_t) \times \Psi({}_R X_t)} \right) \equiv +1^2 \quad (136)$$

is also true, the absence of any technical errors and other errors of human reasoning presupposed. The starting point of this proof (premise:  $+1 = +1$ ) is true. Multiplying Eq. 135 by  ${}_R X_t$ , it is

$${}_R X_t = {}_R X_t \quad (137)$$

Based on theorem 2.7, Eq. 123, Eq. 137 changes to

$$E({}_R X_t) + E({}_R \underline{X}_t) \equiv {}_R X_t \quad (138)$$

Theoretically it is necessary to consider the possibility that there are conditions where  ${}_R X_t$  is in a state of superposition of  $E({}_R X_t)$  and  $E({}_R \underline{X}_t)$ . Thus far, under conditions where Eq. 138 can be described by a (linear) function  $\Psi({}_R X_t)$  which satisfies the superposition principle, it is equally

$$\Psi(E({}_R X_t)) + \Psi(E({}_R \underline{X}_t)) \equiv \Psi(E({}_R X_t) + E({}_R \underline{X}_t)) \equiv \Psi({}_R X_t) \quad (139)$$

The principle of superposition and the Pythagorean theorem are the two sides of the same coin. It is

$$\Psi(E({}_R X_t)) + \Psi(E({}_R \underline{X}_t)) \equiv \Psi({}_R X_t) \quad (140)$$

Normalizing the relationship before, Eq. 140 changes slightly. It is

$$\frac{\Psi(E({}_R X_t))}{\Psi({}_R X_t)} + \frac{\Psi(E({}_R \underline{X}_t))}{\Psi({}_R X_t)} \equiv \frac{\Psi({}_R X_t)}{\Psi({}_R X_t)} \equiv +1^1 \quad (141)$$

Multiplying Eq. 141 by  $\left( \frac{\Psi({}_R X_t)}{\Psi({}_R X_t)} \right)$  it is,

$$\left( \frac{\Psi({}_R X_t) \times \Psi(E({}_R X_t))}{\Psi({}_R X_t) \times \Psi({}_R X_t)} \right) + \left( \frac{\Psi({}_R X_t) \times \Psi(E({}_R \underline{X}_t))}{\Psi({}_R X_t) \times \Psi({}_R X_t)} \right) \equiv +1^2 \quad (142)$$

□

**Theorem 2.10 (THE GENERAL CONTRADICTION LAW).** *In general, it is*

$$E(\underline{R}X_t) \leq \left( \frac{{}_R X_t^2 \times \pi^2 \times \hbar^2}{E({}_R X_t) \times h^2} \right) \quad (143)$$

*Proof.* If the premise

$$+1 = +1 \quad (144)$$

is true, **then** the conclusion

$$E(\underline{R}X_t) \leq \left( \frac{{}_R X_t^2 \times \pi^2 \times \hbar^2}{E({}_R X_t) \times h^2} \right) \quad (145)$$

is also true, the absence of any technical errors and other errors of human reasoning presupposed. The starting point of this proof (premise:  $+1 = +1$ ) is true. Multiplying Eq. 144 by the variance of  ${}_R X_t$  denoted as  $\sigma({}_R X_t)^2$  (see also Kolmogorov, Andreï Nikolaevich, 1950, p. 42), it is

$$\sigma({}_R X_t)^2 \equiv \sigma({}_R X_t)^2 \quad (146)$$

The variance of  ${}_R X_t$ , denoted as  $\sigma({}_R X_t)^2$  (see also Kolmogorov, Andreï Nikolaevich, 1950, p. 42), is defined or has been proved as

$$\sigma({}_R X_t)^2 \equiv E({}_R X_t) \times E(\underline{R}X_t) \quad (147)$$

In general, according to theorem 2.3, Eq. 49, it is

$$p({}_R X_t) \equiv \frac{E({}_R X_t)}{{}_R X_t} \quad (148)$$

while theorem 2.3, Eq. 50 demands that

$$p(\underline{R}X_t) \equiv \frac{E(\underline{R}X_t)}{{}_R X_t} = 1 - \frac{E({}_R X_t)}{{}_R X_t} = 1 - p({}_R X_t) \quad (149)$$

Therefore, Eq. 147 changes to

$$\begin{aligned} \sigma({}_R X_t)^2 &\equiv \sigma({}_R X_t) \times \sigma(\underline{R}X_t) \\ &\equiv E({}_R X_t - E({}_R X_t))^2 \\ &\equiv ({}_R X_t^2) \times (p({}_R X_t) \times (1 - (p({}_R X_t)))) \\ &\equiv E({}_R X_t) \times E(\underline{R}X_t) \end{aligned} \quad (150)$$

Eq. 150 simplifies as

$$\begin{aligned} \sigma({}_R X_t)^2 &\equiv E({}_R X_t) \times E(\underline{R}X_t) \\ &\equiv ({}_R X_t^2) \times (p({}_R X_t) \times (1 - (p({}_R X_t)))) \end{aligned} \quad (151)$$

Under conditions, where the probability of a single event is not known, it is

$$(p({}_R X_t) \times (1 - (p({}_R X_t)))) \leq \frac{1}{4} \quad (152)$$

Eq. 151 changes slightly to

$$\frac{E(\mathcal{R}X_t) \times E(\mathcal{R}\underline{X}_t)}{\mathcal{R}X_t^2} \leq \left(\frac{1}{2} \times \frac{1}{2}\right) \quad (153)$$

From quantum theory, it is known that

$$\frac{1}{2} \equiv \frac{\pi \times \hbar}{h} \quad (154)$$

Eq. 153 changes to

$$\frac{E(\mathcal{R}X_t) \times E(\mathcal{R}\underline{X}_t)}{\mathcal{R}X_t^2} \leq \left(\frac{\pi^2 \times \hbar^2}{h^2}\right) \quad (155)$$

The expectation value of anti  $\mathcal{R}X_t$ , denoted as  $E(\mathcal{R}\underline{X}_t)$ , follows approximately as

$$E(\mathcal{R}\underline{X}_t) \leq \left(\frac{\mathcal{R}X_t^2 \times \pi^2 \times \hbar^2}{E(\mathcal{R}X_t) \times h^2}\right) \quad (156)$$

□

Eq. 156 does not give any reason for the assumption that there is a kind of uncertainty between  $\mathcal{R}X_t$  and  $\mathcal{R}\underline{X}_t$  and do not constitute in no way a new uncertainty principle. Under conditions of 4 space-time dimensions of general relativity, it is

$$\frac{1}{g_{\mu\nu} \times g^{\mu\nu}} \equiv \frac{1}{4} \quad (157)$$

Eq. 153 changes under these conditions of general relativity to

$$\frac{E(\mathcal{R}X_t) \times E(\mathcal{R}\underline{X}_t)}{\mathcal{R}X_t^2} \leq \frac{1}{g_{\mu\nu} \times g^{\mu\nu}} \quad (158)$$

or to

$$\mathcal{R}X_t^2 \geq E(\mathcal{R}X_t) \times g_{\mu\nu} \times E(\mathcal{R}\underline{X}_t) \times g^{\mu\nu} \quad (159)$$

Furthermore, under conditions where

$$E(\mathcal{R}X_t) + E(\mathcal{R}\underline{X}_t) \equiv \mathcal{R}X_t \quad (160)$$

we obtain, the identity (see also Barukčić, 2020a,b, 2021c) of

$$\mathcal{R}\Delta_t^2 \equiv \sigma(\mathcal{R}X_t)^2 \quad (161)$$

Especially, general relativity is related to the Pythagorean theorem. General relativity is a theory of the geometrical properties of space-time to, while the metric tensor  $g_{\mu\nu}$  itself is of fundamental importance for general relativity. An important differentiation with respect to the metric tensor  $g_{\mu\nu}$  is necessary. The metric tensor  $g_{\mu\nu}$  does not describe above all the gravitational field, but the gravitational potential. Einstein himself worded this fact excellently.

“... die ... Komponenten des Gravitationspotentials  $g_{\mu\nu}$  ... ”

(see also Einstein, 1916, p. 818)

In English: ‘... the ... components of the gravitational potential  $g_{\mu\nu}$  ...’. The metric tensor  $g_{\mu\nu}$  is something like the generalization of the Pythagorean theorem. Thus far, it does not appear to be necessary to restrict the validity of the Pythagorean theorem only to certain situations. The question is justified why the Riemannian geometry should be oppressed by the quadratic restriction. In this context, **Finsler geometry**, named after Paul Finsler (1894 - 1970) who studied it in his doctoral thesis (see Finsler, 1918) in 1918, appears to be a kind of metric generalization of Riemannian geometry without the quadratic restriction and justifies the attempt to systematize and to extend the possibilities of general relativity.

### 2.2.3. A circle and a right-angled triangle

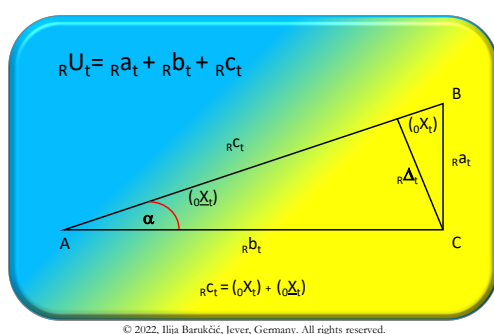
**2.2.3.1. The equivalence of a circle and a right-angled triangle** Let  ${}_R U_t$  denote the circumference of a circle (see Book 3 of Euclid’s Elements). It is

$${}_R U_t \equiv 2 \times \pi \times {}_R r_t \equiv \pi \times {}_R d_t \quad (162)$$

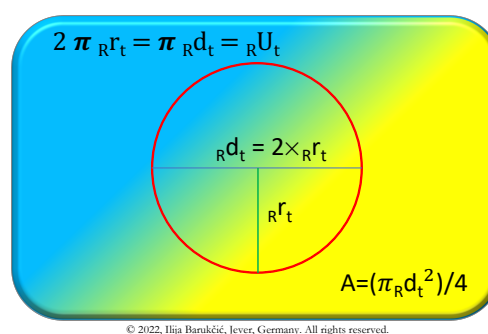
Let,

$${}_R d_t \equiv 2 \times {}_R r_t \quad (163)$$

where  ${}_R d_t$  is the diameter of a circle and  ${}_R r_t$  is the radius of a circle, the distance between any point of a circle and the centre of the same circle.



**Figure 6. Circumference: right-angled triangle**



**Figure 7. Circumference: circle**

As can be seen, the circumference of a right-angled triangle (see figure 6) is given as

$${}_R U_t \equiv {}_R a_t + {}_R b_t + {}_R c_t \quad (164)$$

However, in nature, under conditions where a circle passes over into a right-angled triangle and vice versa (see figure 6 and figure 7) it is

$${}_R U_t \equiv {}_R a_t + {}_R b_t + {}_R c_t \equiv 2 \times \pi \times {}_R r_t \equiv \pi \times {}_R d_t \equiv {}_R U_t \quad (165)$$

It is known that

$${}_R c_t \equiv \sqrt{{}_R a_t^2 + {}_R b_t^2} \quad (166)$$

Equation 165 becomes

$${}_R U_t \equiv ({}_R a_t) + ({}_R b_t) + \sqrt{{}_R a_t^2 + {}_R b_t^2} \equiv \pi \times {}_R d_t \quad (167)$$

Max Karl Ernst Ludwig Planck related mass to frequency and introduced  $h$ , the famous Planck's constant (see also Planck, 1901, p. 87). Soon, Dirac adopted Planck's constant  $h$ . "In Order that the theory may agree with experiment, we must take  $\hbar$  equal to  $h/2\pi$ , where  $h$  is the universal constant that was introduced by Planck, known as Planck's constant." (see also Dirac, 1947, p. 87) or

$$h = 2 \times \pi \times \hbar \quad (168)$$

Archimedes constant  $\pi$ , approximately equal to 3.1415926535897932384626433..., can be calculated as

$$\pi \equiv \frac{{}_R U_t}{{}_R d_t} \equiv \frac{({}_R a_t) + ({}_R b_t) + \sqrt{{}_R a_t^2 + {}_R b_t^2}}{{}_R d_t} \equiv \frac{h}{2 \times \hbar} \quad (169)$$

while the diameter of a circle,  ${}_R d_t$ , is given as

$${}_R d_t \equiv \frac{{}_R U_t}{\pi} \equiv \frac{({}_R a_t) + ({}_R b_t) + \sqrt{{}_R a_t^2 + {}_R b_t^2}}{\pi} \equiv \frac{({}_R a_t) + ({}_R b_t) + \sqrt{{}_R a_t^2 + {}_R b_t^2}}{\frac{h}{2 \times \hbar}} \quad (170)$$

Planck's constant  $h$  (see equation 169) appears to be very dynamical and can be calculated as

$$h \equiv 2 \times \frac{({}_R a_t) + ({}_R b_t) + \sqrt{{}_R a_t^2 + {}_R b_t^2}}{{}_R d_t} \times \hbar \quad (171)$$

Especially under conditions where  $({}_R a_t) \equiv \sqrt[2]{p({}_R X_t)}$  and where  $({}_R b_t) \equiv \sqrt[2]{p({}_R X_t)}$  Planck's constant becomes

$$h \equiv 2 \times \frac{\sqrt[2]{p({}_R X_t)} + \sqrt[2]{p({}_R X_t)} + 1}{{}_R d_t} \times \hbar \quad (172)$$

**2.2.3.2.  $\pi$  and trigonometry** In Euclidean geometry, the number  $\pi = 3.14159 \dots$ , also referred to as Archimedes's constant, is defined as the ratio of a circle's circumference  ${}_R U_t$  to its diameter  ${}_R d_t$ . The first (see Jones, William, 1706, p. 263) known use of the Greek letter  $\pi$  to represent the ratio of a circle's circumference  ${}_R U_t$  to its diameter  ${}_R d_t$  is ascribed to the Welsh mathematician William Jones (1675 – 1749) in 1706.



**Theorem 2.11.** *In general, it is*

$${}_R U_t \equiv ((\sin \alpha) + (\cos \alpha) + 1) \times {}_R C_t \quad (173)$$

*Proof by direct proof.* The premise

$$+1 \equiv +1 \quad (174)$$

is true. In the following, we rearrange the premise. We obtain (see equation 166, p. 44)

$$\begin{aligned} {}_R U_t &\equiv {}_R a_t + {}_R b_t + {}_R c_t \\ &\equiv 2 \times \pi \times {}_R r_t \\ &\equiv \pi \times {}_R d_t \\ &\equiv \pi \times X \times {}_R C_t \\ &\equiv {}_R U_t \end{aligned} \quad (175)$$

Equation 175 is rearranged as

$${}_R a_t + {}_R b_t + {}_R c_t \equiv \pi \times X \times {}_R C_t \quad (176)$$

and changes to

$$\frac{{}_R a_t}{{}_R C_t} + \frac{{}_R b_t}{{}_R C_t} + \frac{{}_R c_t}{{}_R C_t} \equiv \pi \times X \quad (177)$$

It is  $\sin \alpha \equiv \frac{{}_R a_t}{{}_R C_t}$  and  $\cos \alpha \equiv \frac{{}_R b_t}{{}_R C_t}$  and  $\frac{{}_R c_t}{{}_R C_t} \equiv +1$ . Equation 177 simplifies as

$$((\sin \alpha) + (\cos \alpha) + 1) \equiv \pi \times X \quad (178)$$

The unknown value of X follows as

$$X \equiv \frac{\sin \alpha}{\pi} + \frac{\cos \alpha}{\pi} + \frac{1}{\pi} \quad (179)$$

The circle's circumference  ${}_R U_t$  is given as (see equation 175, p. 45)

$$\begin{aligned} {}_R U_t &\equiv \pi \times {}_R d_t \\ &\equiv 2 \times \pi \times {}_R r_t \\ &\equiv \pi \times X \times {}_R C_t \\ &\equiv \pi \times \left( \frac{\sin \alpha}{\pi} + \frac{\cos \alpha}{\pi} + \frac{1}{\pi} \right) \times {}_R C_t \\ &\equiv \pi \times \left( \frac{(\sin \alpha) + (\cos \alpha) + 1}{\pi} \right) \times {}_R C_t \end{aligned} \quad (180)$$

and finally as

$${}_R U_t \equiv ((\sin \alpha) + (\cos \alpha) + 1) \times {}_R C_t \quad (181)$$

□

Based on equation 181,  $\pi$  is given as the relationship

$$\pi \equiv \frac{{}_R U_t}{{}_R d_t} \equiv \frac{((\sin \alpha) + (\cos \alpha) + 1) \times {}_R c_t}{{}_R d_t} \quad (182)$$

#### 2.2.4. Bernoulli distribution

A single event distribution is more or less a discrete probability distribution of any random variable  $X$  which takes a certain (observer independent) single value  $X_t$  at a **Bernoulli trial** (Uspensky, 1937, p. 45) (period of time)  $t$  with the probability  $p(X_t)$ . The same random variable  $X$  takes a certain single anti value  $\underline{X}_t$  at a Bernoulli trial (period of time)  $t$  with the probability  $1-p(X_t)$ . There are conditions in nature where a random variable  $X$  can take only the values either  $+0$  or  $+1$  (see Birnbaum, 1961). Under these conditions, the random variable  $X$  takes the value 1 with probability  $p(X_t = +1)$  and the value 0 with probability  $q(X_t = +0) = 1 - p(X_t = +1)$  while the single event distribution passes over into the **Bernoulli distribution**, named after Swiss mathematician Jacob Bernoulli (Bernoulli, 1713). Less formally, many times, the Bernoulli distribution is represented by a (possibly not biased) coin toss where 1 and 0 would represent ‘heads’ and ‘tails’ (or vice versa), respectively. However, the relationship between random variables (Gosset, 1914) can be investigated by many (Gosset, 1908) methods, including the tools of probability theory, too.

#### Definition 2.11 (Two by two table of single event random variables).

The two by two or contingency table which has been introduced by Karl Pearson (Pearson, 1904b) in 1904 harbours still a large variety of topics and debates. Central to this is the problem to apply the laws of classical logic on data sets, which concerns the justification of inferences which extrapolate from sample data to general facts. Nevertheless, a contingency table is still an appropriate theoretical model too for studying the relationships between random variables, including *Bernoulli* (Bernoulli, 1713) (i.e.  $+0/+1$ ) distributed random variables existing or occurring at the same *Bernoulli trial* (Uspensky, 1937) (period of time)  $t$ .

In this context, let a random variable  $A$  at the *Bernoulli trial* (Uspensky, 1937) (period of time)  $t$ , denoted by  $A_t$ , indicate a risk factor, a condition, a cause et cetera and occur or exist with the probability  $p(A_t)$  at the *Bernoulli trial* (Uspensky, 1937) (period of time)  $t$ . Let  $E(A_t)$  denote the expectation value of  $A_t$ . In general it is

$$p(A_t) \equiv p(a_t) + p(b_t) \quad (183)$$

The expectation value  $E(A_t)$  follows as

$$\begin{aligned} E(A_t) &\equiv A_t \times p(A_t) \\ &\equiv A_t \times (p(a_t) + p(b_t)) \\ &\equiv (A_t \times p(a_t)) + (A_t \times p(b_t)) \\ &\equiv E(a_t) + E(b_t) \end{aligned} \quad (184)$$

Under conditions of +0/+1 distributed Bernoulli random variables it is

$$\begin{aligned}
 E(A_t) &\equiv A_t \times p(A_t) \\
 &\equiv (+0 + 1) \times p(A_t) \\
 &\equiv p(A_t) \\
 &\equiv p(a_t) + p(b_t)
 \end{aligned}
 \tag{185}$$

Furthermore, it is

$$p(\underline{A}_t) \equiv p(c_t) + p(d_t) \equiv (1 - p(A_t)) \tag{186}$$

The expectation value  $E(\underline{A}_t)$  is given as

$$\begin{aligned}
 E(\underline{A}_t) &\equiv A_t \times (1 - p(A_t)) \\
 &\equiv A_t \times (p(c_t) + p(d_t)) \\
 &\equiv (A_t \times p(c_t)) + (A_t \times p(d_t)) \\
 &\equiv E(c_t) + E(d_t)
 \end{aligned}
 \tag{187}$$

Under conditions of +0/+1 distributed Bernoulli random variables we obtain

$$\begin{aligned}
 E(\underline{A}_t) &\equiv A_t \times (1 - p(A_t)) \\
 &\equiv (+0 + 1) \times (1 - p(A_t)) \\
 &\equiv (1 - p(A_t)) \\
 &\equiv p(c_t) + p(d_t)
 \end{aligned}
 \tag{188}$$

Let a random variable  $B$  at the *Bernoulli trial* (Uspensky, 1937) (period of time)  $t$ , denoted by  $B_t$ , indicate an outcome, a conditioned, an effect et cetera and occur or exist with the probability  $p(B_t)$  at the *Bernoulli trial* (Uspensky, 1937) (period of time)  $t$ . Let  $E(B_t)$  denote the expectation value of  $B_t$ . In general it is

$$p(B_t) \equiv p(a_t) + p(c_t) \tag{189}$$

The expectation value  $E(B_t)$  is given by the equation

$$\begin{aligned}
 E(B_t) &\equiv B_t \times p(B_t) \\
 &\equiv B_t \times (p(a_t) + p(c_t)) \\
 &\equiv (B_t \times p(a_t)) + (B_t \times p(c_t)) \\
 &\equiv E(a_t) + E(c_t)
 \end{aligned}
 \tag{190}$$

Under conditions of +0/+1 distributed Bernoulli random variables it is

$$\begin{aligned}
 E(B_t) &\equiv B_t \times p(B_t) \\
 &\equiv (+0 + 1) \times p(B_t) \\
 &\equiv p(B_t) \\
 &\equiv p(a_t) + p(c_t)
 \end{aligned}
 \tag{191}$$

Furthermore, it is

$$p(\underline{B}_t) \equiv p(b_t) + p(d_t) \equiv (1 - p(B_t)) \tag{192}$$

The expectation value  $E(\underline{B}_t)$  is given by the equation

$$\begin{aligned}
 E(\underline{B}_t) &\equiv B_t \times (1 - p(B_t)) \\
 &\equiv B_t \times (p(b_t) + p(d_t)) \\
 &\equiv (B_t \times p(b_t)) + (B_t \times p(d_t)) \\
 &\equiv E(b_t) + E(d_t)
 \end{aligned} \tag{193}$$

Under conditions of +0/+1 distributed Bernoulli random variables it is

$$\begin{aligned}
 E(\underline{B}_t) &\equiv B_t \times (1 - p(B_t)) \\
 &\equiv (+0 + 1) \times (1 - p(B_t)) \\
 &\equiv (1 - p(B_t)) \\
 &\equiv p(b_t) + p(d_t)
 \end{aligned} \tag{194}$$

Let  $p(a_t) = p(A_t \wedge B_t)$  denote the joint probability distribution of  $A_t$  and  $B_t$  at the same Bernoulli trial (period of time)  $t$ . In general, it is

$$\begin{aligned}
 E(a_t) &\equiv E(A_t \wedge B_t) \\
 &\equiv (A_t \times B_t) \times p(A_t \wedge B_t) \\
 &\equiv (A_t \times B_t) \times p(a_t)
 \end{aligned} \tag{195}$$

Under conditions of +0/+1 distributed Bernoulli random variables, it is

$$\begin{aligned}
 E(a_t) &\equiv E(A_t \wedge B_t) \\
 &\equiv (A_t \times B_t) \times p(A_t \wedge B_t) \\
 &\equiv ((+0 + 1) \times (+0 + 1)) \times p(A_t \wedge B_t) \\
 &\equiv p(A_t \wedge B_t) \\
 &\equiv p(a_t)
 \end{aligned} \tag{196}$$

Let  $p(b_t) = p(A_t \wedge \neg B_t)$  denote the joint probability distribution of  $A_t$  and not  $B_t$  at the same Bernoulli trial (period of time)  $t$ . In general, it is

$$\begin{aligned}
 E(b_t) &\equiv E(A_t \wedge \neg B_t) \\
 &\equiv (A_t \times \neg B_t) \times p(A_t \wedge \neg B_t) \\
 &\equiv (A_t \times \neg B_t) \times p(b_t)
 \end{aligned} \tag{197}$$

Under conditions of +0/+1 distributed Bernoulli random variables, it is

$$\begin{aligned}
 E(b_t) &\equiv E(A_t \wedge \neg B_t) \\
 &\equiv (A_t \times \neg B_t) \times p(A_t \wedge \neg B_t) \\
 &\equiv ((+0 + 1) \times (+0 + 1)) \times p(A_t \wedge \neg B_t) \\
 &\equiv p(A_t \wedge \neg B_t) \\
 &\equiv p(b_t)
 \end{aligned} \tag{198}$$

Let  $p(c_t) = p(\neg A_t \wedge B_t)$  denote the joint probability distribution of not  $A_t$  and  $B_t$  at the same Bernoulli trial (period of time)  $t$ . In general, it is

$$\begin{aligned} E(c_t) &\equiv E(\neg A_t \wedge B_t) \\ &\equiv (\neg A_t \wedge B_t) \times p(\neg A_t \wedge B_t) \\ &\equiv (\neg A_t \wedge B_t) \times p(c_t) \end{aligned} \quad (199)$$

Under conditions of +0/+1 distributed Bernoulli random variables, it is

$$\begin{aligned} E(c_t) &\equiv E(\neg A_t \wedge B_t) \\ &\equiv (\neg A_t \times B_t) \times p(\neg A_t \wedge B_t) \\ &\equiv ((+0 + 1) \times (+0 + 1)) \times p(\neg A_t \wedge B_t) \\ &\equiv p(\neg A_t \wedge B_t) \\ &\equiv p(c_t) \end{aligned} \quad (200)$$

Let  $p(d_t) = p(\neg A_t \wedge \neg B_t)$  denote the joint probability distribution of not  $A_t$  and not  $B_t$  at the same Bernoulli trial (period of time)  $t$ . In general, it is

$$\begin{aligned} E(d_t) &\equiv E(\neg A_t \times \neg B_t) \\ &\equiv (\neg A_t \times \neg B_t) \times p(\neg A_t \wedge \neg B_t) \\ &\equiv (\neg A_t \times \neg B_t) \times p(d_t) \end{aligned} \quad (201)$$

Under conditions of +0/+1 distributed Bernoulli random variables, it is

$$\begin{aligned} E(d_t) &\equiv E(\neg A_t \wedge \neg B_t) \\ &\equiv (\neg A_t \times \neg B_t) \times p(\neg A_t \wedge \neg B_t) \\ &\equiv ((+0 + 1) \times (+0 + 1)) \times p(\neg A_t \wedge \neg B_t) \\ &\equiv p(\neg A_t \wedge \neg B_t) \\ &\equiv p(d_t) \end{aligned} \quad (202)$$

In general, it is

$$p(a_t) + p(b_t) + p(c_t) + p(d_t) \equiv +1 \quad (203)$$

Table 2 provide us with an overview of the definitions above.

**Table 2.** The two by two table of Bernoulli random variables

		Conditioned $B_t$		
		TRUE	FALSE	
Condition $A_t$	TRUE	$p(a_t)$	$p(b_t)$	$p(A_t)$
	FALSE	$p(c_t)$	$p(d_t)$	$p(\underline{A}_t)$
		$p(B_t)$	$p(\underline{B}_t)$	+1

In our understanding, it is

$$p(B_t) + p(\Lambda_t) \equiv p(a_t) + p(c_t) + p(\Lambda_t) \equiv p(a_t) + p(b_t) \equiv p(A_t) \quad (204)$$

or

$$p(c_t) + p(\Lambda_t) \equiv p(b_t) \quad (205)$$

Under conditions of Einstein's general theory of relativity,  $\Lambda$  denotes the Einstein cosmological (Einstein, 1917) 'constant'.

### 2.2.5. Binomial and Anti-binomial distribution

The binomial (see Pearson, 1895, p. 351) distribution (see Cramér, 1937) with parameters  $n$  and  $p$  has been developed by the Swiss mathematician Jakob Bernoulli (1655-1705) in a proof published in his 1713 book *Ars Conjectandi* (see Bernoulli, 1713) Part 1. In probability theory and statistics, the probability of getting exactly  $k$  successes in  $n$  independent Bernoulli trials is given by the probability mass function as

$$p(X_t = k) \equiv \binom{n}{k} \times p^k \times q^{n-k} \quad (206)$$

while is  $\binom{n}{k} = \frac{n!}{k!(n-k)!}$  the binomial coefficient. The Anti-binomial distribution, the other or the complementary of a Binomial distribution, denoted as  $p(X_t = \underline{k})$  is given as:

$$p(X_t = \underline{k}) = 1 - p(X_t = k) \equiv 1 - \binom{n}{k} \times p^k \times q^{n-k} \quad (207)$$

The variance of a Binomial distributed event is given as

$$\begin{aligned} \sigma(k)^2 &\equiv k \times k \times p(k) \times p(\underline{k}) \\ &\equiv (k) \times (k) \times \left( \binom{n}{k} \times p^k \times q^{n-k} \right) \times \left( 1 - \left( \binom{n}{k} \times p^k \times q^{n-k} \right) \right) \end{aligned} \quad (208)$$

The relationship between Binomial distribution and Anti-binomial distribution is illustrated by fig. 8 and fig. 9 in more detail.

As known, the cumulative distribution function is given as

$$p(X_t \leq k) \equiv 1 - p(X_t > k) \equiv \sum_{t=0}^k \binom{n}{t} \cdot p^t \cdot q^{n-t} \quad (209)$$

or as

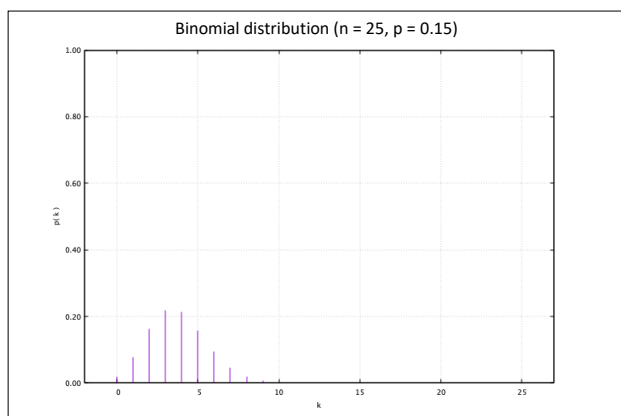
$$p(X_t > k) \equiv 1 - p(X_t \leq k) \equiv 1 - \sum_{t=0}^k \binom{n}{t} \cdot p^t \cdot q^{n-t} \quad (210)$$

Furthermore, it is

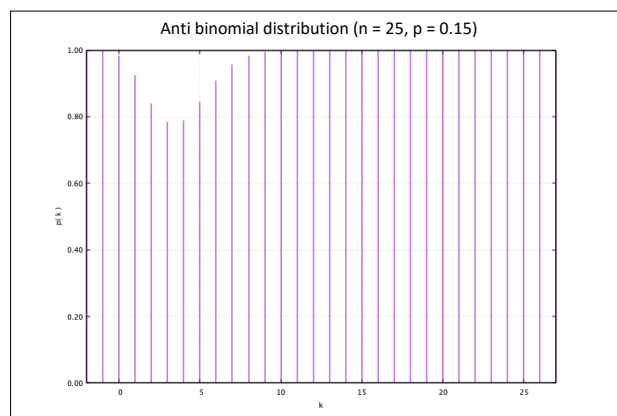
$$p(X_t < k) \equiv 1 - p(X_t \geq k) \equiv \sum_{t=0}^{k-1} \binom{n}{t} \cdot p^t \cdot q^{n-t} \quad (211)$$

or

$$p(X_t \geq k) \equiv 1 - p(X_t < k) \equiv 1 - \sum_{t=0}^{k-1} \binom{n}{t} \cdot p^t \cdot q^{n-t} \quad (212)$$



**Figure 8. Binomial distribution.**



**Figure 9. Anti-binomial dis.**

The binomial distribution is the mathematical foundation of a binomial test. The random variable  $X_t$  is counting for different things. The discrete geometric (see [Feller, 1950](#), p. 61) distribution describes under certain circumstances the number of Bernoulli trials needed to get one success. The probability that the first occurrence of success requires  $k$  independent trials, each with success probability  $p$ , is given by the equation

$$p(X_t = k) \equiv p \cdot q^{k-1} \quad (213)$$

The negative (see [Fisher, 1941](#), [Haldane, 1941](#)) binomial probability is a discrete probability distribution which defines the number of successes ( $k$ ) in a sequence of independent and identically distributed Bernoulli trials ( $n$ ) before a specified (non-random) number of failures (denoted  $r$ ) occurs. The probability mass function of the negative binomial distribution is

$$p(X_t = r) \equiv \binom{k+r-1}{k-1} p^k \cdot q^r \quad (214)$$

where  $k$  is the number of successes,  $r$  is the number of failures, and  $p$  is the probability of success.

**Definition 2.12 (Expectation value and variance of a binomial random variable).**

The variance (see [Pearson, 1904a](#), p. 66) of a binomial distributed random variable with parameters  $n$ , the number of independent experiments each asking a yes–no question and  $p$ , the probability of a single event, is defined in contrast to Pearson (see [Barukčić, 2022c](#)) as

$$\sigma(X_t)^2 \equiv N \times N \times p(X_t) \times (1 - p(X_t)) \quad (215)$$

**Definition 2.13 (Two by two table of Binomial random variables).**

Let  $a$ ,  $b$ ,  $c$ ,  $d$ ,  $\underline{A}$ ,  $\underline{B}$ , and  $\underline{B}$  denote expectation values. Under conditions where *the probability of an event, an outcome, a success et cetera is constant from Bernoulli trial to Bernoulli trial  $t$* , it is

$$\begin{aligned}
 A &= N \times E(A_t) \\
 &\equiv N \times (A_t \times p(A_t)) \\
 &\equiv N \times (p(A_t) + p(B_t)) \\
 &\equiv N \times p(A_t)
 \end{aligned}
 \tag{216}$$

and

$$\begin{aligned}
 B &= N \times E(B_t) \\
 &\equiv N \times (B_t \times p(B_t)) \\
 &\equiv N \times (p(A_t) + p(c_t)) \\
 &\equiv N \times p(B_t)
 \end{aligned}
 \tag{217}$$

where  $N$  might denote the population or even the sample size. Furthermore, it is

$$a \equiv N \times (E(A_t)) \equiv N \times (p(A_t)) \tag{218}$$

and

$$b \equiv N \times (E(B_t)) \equiv N \times (p(B_t)) \tag{219}$$

and

$$c \equiv N \times (E(c_t)) \equiv N \times (p(c_t)) \tag{220}$$

and

$$d \equiv N \times (E(d_t)) \equiv N \times (p(d_t)) \tag{221}$$

and

$$a + b + c + d \equiv A + \underline{A} \equiv B + \underline{B} \equiv N \tag{222}$$

Table 3 provide us again an overview of a two by two contingency (see also [Pearson, 1904b](#), p. 33) table of Binomial random variables.

“Such a table is termed a contingency table, and the ultimate scientific statement of description of the relation between two things can always be thrown back upon such a contingency table . . . Once the reader realizes the nature of such a table, he will have grasped the essence of the conception of association between cause and effect, and the nature of its ideal limit in causation. ”

(see also [Pearson, 1911](#), p. 159)



**Table 3.** The two by two table of Binomial random variables

		Conditioned $B_t$		
		TRUE	FALSE	
Condition	TRUE	a	b	A
	FALSE	c	d	<u>A</u>
		B	<u>B</u>	N

### 2.2.6. Poisson and Anti-Poisson distribution

The Poisson distribution (see also [Poisson, 1829](#), pp. 261-262) is a discrete distribution (with  $k = 0, 1, 2, 3, \dots$ ) which depends on  $\lambda$ , the mean number of occurrences expected (see also [Poisson and Poisson, 1837](#), pp. 205) while there is no specified number  $n$  of possible tries. The probability of a given number of events occurring in a fixed interval of time or space under the condition that these events occur with a known constant mean rate and independently of the time since the last event, is calculated as

$$p(k) = \left(\frac{\lambda^k}{k!}\right) \times e^{-\lambda} \quad (223)$$

Cavalry men being killed by a kick of a horse (see also [von Bortkiewitsch, 1898](#)) is a famous example of Poisson distribution. The Anti-Poisson distribution, the other of the Poisson distribution or the complementary of the Poisson distribution, denoted as  $p(\underline{k})$  is given as

$$p(\underline{k}) = 1 - p(k) = 1 - \left(\frac{\lambda^k}{k!}\right) \times e^{-\lambda} \quad (224)$$

The variance of a Poisson distributed event is given as

$$\begin{aligned} \sigma(k)^2 &\equiv k \times k \times p(k) \times p(\underline{k}) \\ &\equiv (k) \times (k) \times \left(\left(\frac{\lambda^k}{k!}\right) \times e^{-\lambda}\right) \times \left(1 - \left(\left(\frac{\lambda^k}{k!}\right) \times e^{-\lambda}\right)\right) \end{aligned} \quad (225)$$

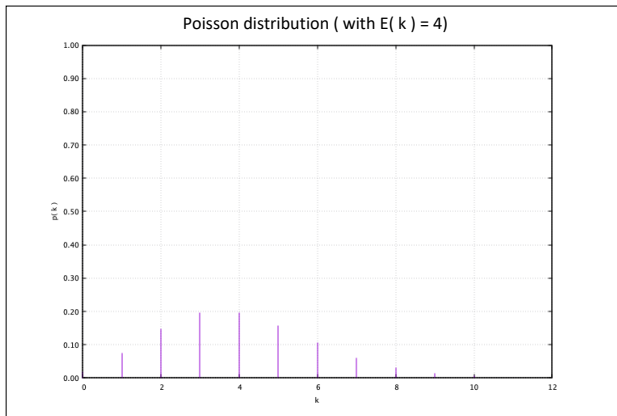
The relationship between the Poisson distribution and the Anti Poisson distribution is illustrated by fig. 10 and fig. 11 in more detail.

### Bombing of London during World War II by Germans

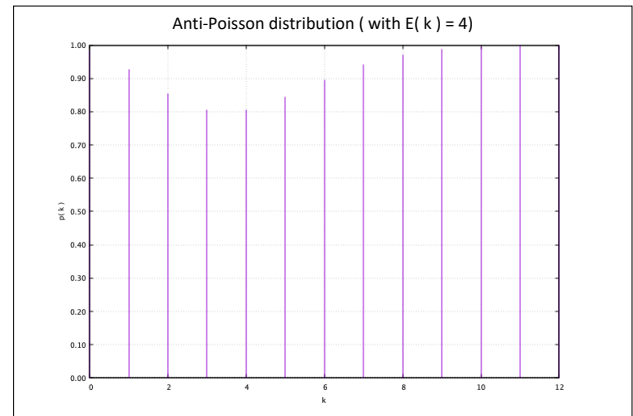
During World War II, London was bombed <sup>92</sup>, <sup>93</sup> by Germans. In order to determine (Clarke, 1946), whether the Germans were bombing London randomly or could target specific areas, London was divided into a grid consisting of 576 equal squares, each square of area 0.25 square kilometres. The number of squares with  $k = 0, 1, \dots$  bombs that landed in each grid square was counted. Over the period considered, the total number of bombs within the area of London involved was 537. The data are illustrated by fig. 4.

<sup>92</sup>Clarke, R. D. (1946). An application of the Poisson distribution. *Journal of the Institute of Actuaries*, 72(3), 481-481.

<sup>93</sup>Clarke, R. D. (1946). An application of the Poisson distribution. *Journal of the Institute of Actuaries*, 72(3), 481-481.



**Figure 10. Poisson distribution.**



**Figure 11. Anti-poisson dis.**

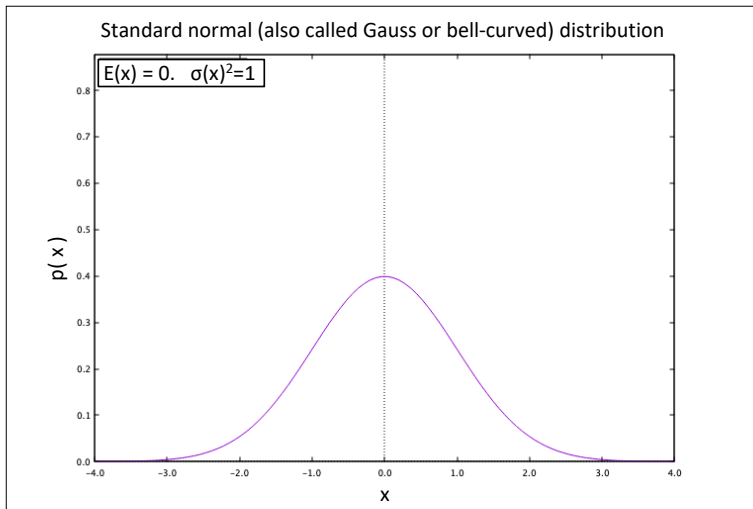
**Table 4. Bombing of London during World War II by Germans**

Number of flying bombs per square (k)	Observed number of squares	Expected number of squares (Poisson)
0	229	226.74
1	211	211.39
2	93	98.54
3	35	30.62
4	7	7.14
5 and over	1	1.57
	576	576

The closeness of fit with the Poisson distribution is obvious and has been tested by the  $\tilde{\chi}^2$  goodness of fit test.

### 2.2.7. Normal and Anti-normal distribution

The origins of the normal distribution, also known as the Gaussian distribution, the second law of Laplace, the law of error et cetera, has been studied at least since the 18th century and can be traced back even to a French mathematician Abraham de Moivre. Johann Carl Friedrich Gauß's (1777-1855) presented 1809 the normal distribution (see [Gauß, Carl Friedrich, 1809](#), p. 244) while illustrating the method of least squares. In the following, Karl Pearson (1857-1936) popularised a new name for Gauß distribution. Pearson wrote: "A frequency-curve, which for practical purposes, can be represented by the error curve, will for the remainder of this paper be termed a normal curve." (see [Pearson, 1894](#), p.



**Figure 12. Normal distribution**

Normal distribution  
with  
 $E(x) = 0$   
and  
 $\sigma(x)^2 = 1$

72).

$$p(\underline{R}X_t) = \left( \frac{1}{\sqrt{2\pi \times \sigma(\underline{R}X_t)^2}} \right) e^{-\frac{(\underline{R}X_t - E(\underline{R}X_t))^2}{2 \times \sigma(\underline{R}X_t)^2}} \quad (226)$$

The standard normal distribution is illustrated by figure 12.

Sir Ronald Aylmer Fisher (1890-1962)<sup>94</sup>, a very influential statistician of the first half of the 20th century, presented the case of a normal (see Fisher, Ronald Aylmer, 1912, p. 157) distribution with non-zero mean (see Fisher, Ronald Aylmer, 1920, p. 758) as a typical case. The probability density function (pdf) of an anti-normal distribution is given as

$$p(\underline{R}X_t) = 1 - \left( \frac{1}{\sqrt{2\pi \times \sigma(\underline{R}X_t)^2}} \right) e^{-\frac{(\underline{R}X_t - E(\underline{R}X_t))^2}{2 \times \sigma(\underline{R}X_t)^2}} \quad (227)$$

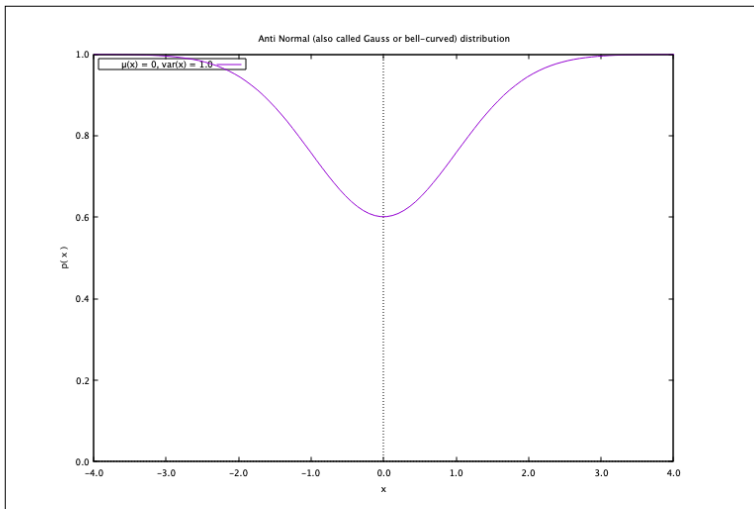
as illustrated by figure 13. In general, it is

$$p(\underline{R}X_t) + p(\underline{R}\underline{X}_t) = 1 \quad (228)$$

The variance of a Gaussian distributed random variable is given as

$$\begin{aligned} \sigma(\underline{R}X_t)^2 &\equiv \underline{R}X_t \times \underline{R}X_t \times p(\underline{R}X_t) \times p(\underline{R}\underline{X}_t) \\ &\equiv \underline{R}X_t \times \underline{R}X_t \times \left( \left( \frac{1}{\sqrt{2\pi \times \sigma(\underline{R}X_t)^2}} \right) e^{-\frac{(\underline{R}X_t - E(\underline{R}X_t))^2}{2 \times \sigma(\underline{R}X_t)^2}} \right) \times \left( 1 - \left( \left( \frac{1}{\sqrt{2\pi \times \sigma(\underline{R}X_t)^2}} \right) e^{-\frac{(\underline{R}X_t - E(\underline{R}X_t))^2}{2 \times \sigma(\underline{R}X_t)^2}} \right) \right) \end{aligned} \quad (229)$$

<sup>94</sup>R. A. Fisher Digital Archive, The University of Adelaide. 5005 AUSTRALIA. copyright@adelaide.edu.au



Anti-normal distribution  
with  
 $E(x) = 0$   
and  
 $\sigma(x)^2 = 1$

**Figure 13. Anti-normal distribution**

Under conditions where  $E({}_R X_t) = 0$  and  $\sigma({}_R X_t)^2 = 1$ , equation 229 becomes

$$\begin{aligned}
 \sigma({}_R X_t)^2 &\equiv {}_R X_t \times {}_R X_t \times p({}_R X_t) \times p({}_R X_t) \\
 &\equiv {}_R X_t \times {}_R X_t \times \left( \left( \frac{1}{\sqrt{2\pi \times \sigma({}_R X_t)^2}} \right) e^{-\frac{({}_R X_t - E({}_R X_t))^2}{2 \times \sigma({}_R X_t)^2}} \right) \times \left( 1 - \left( \left( \frac{1}{\sqrt{2\pi \times \sigma({}_R X_t)^2}} \right) e^{-\frac{({}_R X_t - E({}_R X_t))^2}{2 \times \sigma({}_R X_t)^2}} \right) \right) \\
 &\equiv {}_R X_t \times {}_R X_t \times \left( \left( \frac{1}{\sqrt{2\pi \times 1}} \right) e^{-\frac{({}_R X_t - 0)^2}{2 \times 1}} \right) \times \left( 1 - \left( \left( \frac{1}{\sqrt{2\pi \times 1}} \right) e^{-\frac{({}_R X_t - 0)^2}{2 \times 1}} \right) \right) \\
 &\equiv {}_R X_t \times {}_R X_t \times \left( \left( \frac{1}{\sqrt{2\pi}} \right) e^{-\frac{({}_R X_t)^2}{2}} \right) \times \left( 1 - \left( \left( \frac{1}{\sqrt{2\pi}} \right) e^{-\frac{({}_R X_t)^2}{2}} \right) \right)
 \end{aligned} \tag{230}$$

### Standard normal distribution

In general, a normal distribution with mean 0 and variance 1 is called the standard normal distribution. Modern publications often write the density function for the standard normal distribution, ‘bell-shaped curve’, as

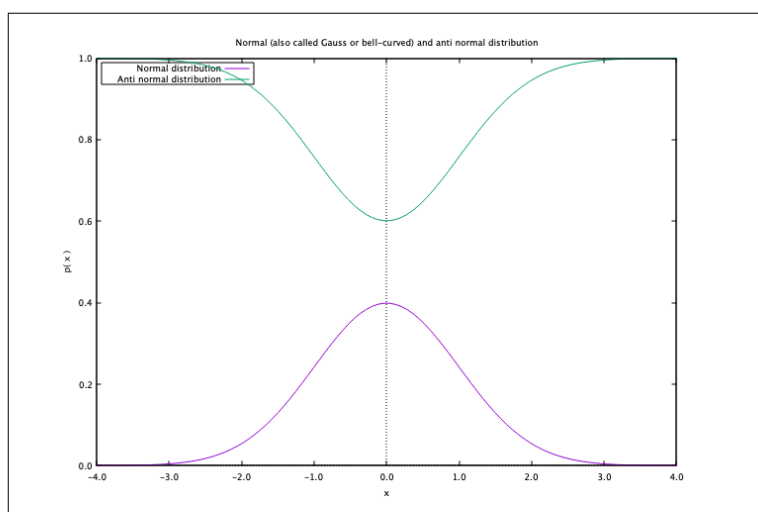
$$p(z) = \left( \frac{1}{\sqrt{2\pi}} \right) e^{-\frac{z^2}{2}} \tag{231}$$

The density function for the anti-standard normal distribution is given as

$$p(\underline{z}) = 1 - p(z) = 1 - \left( \frac{1}{\sqrt{2\pi}} \right) e^{-\frac{z^2}{2}} \tag{232}$$

It is

$$p(\underline{z}) + p(z) = 1 \tag{233}$$



**Figure 14. Normal and anti-normal distribution**

Normal and anti-normal  
distribution  
with  
 $E(x) = 0$   
and  
 $\sigma(x)^2 = 1$

and is illustrated by figure 14.

Truman Lee Kelley (1884–1961) introduced statistical methods into psychological studies<sup>95</sup> and defined the z-score (see Kelley, 1924, p. 115). In mathematical statistics, a random variable  $RX_t$  is standardised by subtracting its expected value  $E(RX_t)$  and dividing the difference by its standard deviation  $\sigma(RX_t)$ . The z-score or standard score, denoted as  $z(RX_t)$ , is defined as

$$z(RX_t) = \frac{(RX_t - E(RX_t))}{\sigma(RX_t)} \quad (234)$$

Simply put, a z-score (also called a standard score) describes how many standard deviations a given quantum mechanical observable or a random variable lies above or below a specific value. Equation 234 changes to

$$z(RX_t)^2 = \frac{(RX_t - E(RX_t))^2}{\sigma(RX_t)^2} = \frac{E(RX_t)^2}{E(RX_t) \times E(RX_t)} = \frac{E(RX_t)}{E(RX_t)} = \frac{RX_t \times (1 - p(RX_t))}{RX_t \times p(RX_t)} = \frac{(1 - p(RX_t))}{p(RX_t)} \quad (235)$$

Equation 235 simplifies as

$$E(RX_t) = z(RX_t)^2 \times E(RX_t) \quad (236)$$

We can imagine drawing figure 14 in  $n$  dimensions. Under these circumstances we would obtain something similar to an **Einstein–Rosen bridge** or Einstein–Rosen wormhole<sup>96</sup> formulated in terms of the framework of probability theory. Attention should be drawn to circumstances especially of quantum mechanics, where  $E(RX_t)$  indicates something like the expectation value of a ‘local hidden variable’. Equation 236 changes slightly. It is

$$RX_t \times (1 - p(RX_t)) = z(RX_t)^2 \times RX_t \times p(RX_t) \quad (237)$$

<sup>95</sup>McClure WE. Speed and Accuracy of the Feebleminded on Performance Tests. *Psychol Clin.* 1931 Feb;19(9):265-274. PMID: 28909304; PMCID: PMC5138284.

<sup>96</sup>Cramer JG, Forward RL, Morris MS, Visser M, Benford G, Landis GA. Natural wormholes as gravitational lenses. *Phys Rev D Part Fields.* 1995 Mar 15;51(6):3117-3120. doi: 10.1103/physrevd.51.3117. PMID: 10018782.

and

$$(1 - p(\mathbb{R}X_t)) = z(\mathbb{R}X_t)^2 \times p(\mathbb{R}X_t) \quad (238)$$

Equation 238 is rearranged as

$$1 = z(\mathbb{R}X_t)^2 \times p(\mathbb{R}X_t) + p(\mathbb{R}X_t) \quad (239)$$

or

$$1 = (z(\mathbb{R}X_t)^2 + 1) \times p(\mathbb{R}X_t) \quad (240)$$

At the end, it follows that

$$p(\mathbb{R}X_t) = \frac{1}{z(\mathbb{R}X_t)^2 + 1} \quad (241)$$

From equation 235 follows that

$$z(\mathbb{R}X_t)^2 = \frac{(\mathbb{R}X_t - E(\mathbb{R}X_t))^2}{\sigma(\mathbb{R}X_t)^2} = \frac{E(\mathbb{R}X_t)^2}{E(\mathbb{R}X_t) \times E(\mathbb{R}X_t)} = \frac{E(\mathbb{R}X_t)}{E(\mathbb{R}X_t)} = \frac{E(\mathbb{R}X_t) \times E(\mathbb{R}X_t)}{E(\mathbb{R}X_t) \times E(\mathbb{R}X_t)} = \frac{\sigma(\mathbb{R}X_t)^2}{E(\mathbb{R}X_t)^2} \quad (242)$$

Thus far, it is equally

$$\sigma(\mathbb{R}X_t)^2 = z(\mathbb{R}X_t)^2 \times E(\mathbb{R}X_t)^2 \quad (243)$$

or

$$\sigma(\mathbb{R}X_t) = z(\mathbb{R}X_t) \times E(\mathbb{R}X_t) \quad (244)$$

Per definition, it is

$$E(\mathbb{R}X_t) = \frac{\sigma(\mathbb{R}X_t)}{z(\mathbb{R}X_t)} \quad (245)$$

The probability density of a halved normal distribution for positive x is given as

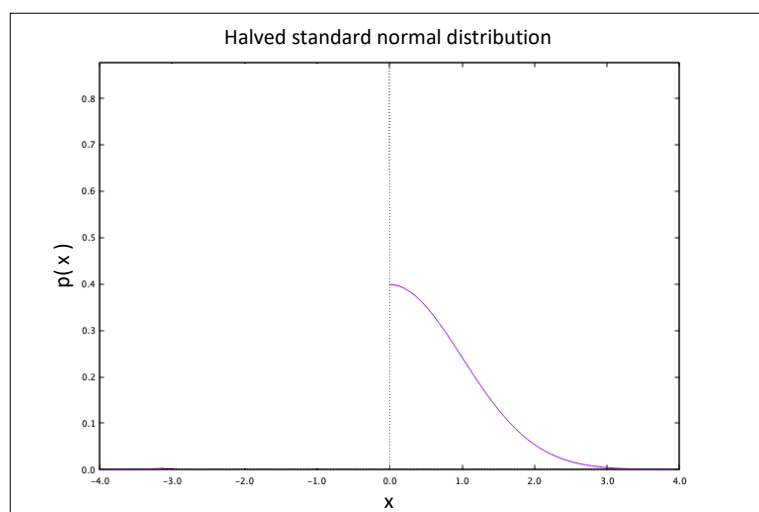
$$p(\mathbb{R}X_t) = \left( \frac{2}{\sqrt{2\pi} \times \sigma(\mathbb{R}X_t)} \right) e^{-\frac{(\mathbb{R}X_t - E(\mathbb{R}X_t))^2}{2 \times \sigma(\mathbb{R}X_t)^2}} \quad (246)$$

and illustrated by figure 15.

## 2.2.8. Independence

### Definition 2.14 (Independence).

The philosophical, mathematical (Kolmogoroff, Andreï Nikolaevich, 1933) and physical (Einstein, 1948) et cetera concept of independence is of fundamental (Kolmogoroff, Andreï Nikolaevich, 1933) importance in (natural) sciences as such. Therefore, it is appropriate to investigate the concept of independence as completely as possible. In fact, de Moivre sums it up in his book *The Doctrine of Chances* (see also Moivre, 1718). “Two Events are **independent**, when they have no connexion one with the other, and that the happening of one neither forwards nor obstructs the happening of the other. Two events are **dependent**, when they are so connected together as that the Probability



**Figure 15. Halved normal distribution**

Halved normal distribution  
with  
 $E(x) = 0$   
and  
 $\sigma(x)^2 = 1$

of either's happening is alter'd by the happening of the other. ”(see also [Moivre, 1756](#), p. 6) We should consider Kolmogorov's position on independence before the mind's eye too. “The concept of mutual independence of two or more experiments holds, in a certain sense, a central position in the theory of probability.”(see also [Kolmogorov, Andreï Nikolaevich, 1950](#), p. 8) Furthermore, it is insightful to recall even Einstein's theoretical approach to the concept of independence. “*Ohne die Annahme einer ... Unabhängigkeit der ... Dinge voneinander ... wäre physikalisches Denken ... nicht möglich.*”(Einstein, 1948). In general, an event  $A_t$  at the Bernoulli trial  $t$  need not, but can be independent of the existence or of the occurrence, of another event  $B_t$  at the same Bernoulli trial  $t$ . De Moivre brings it to the point. “From what has been said, it follows, that if a Fraction expresses the Probability of an Event, and another Fraction the Probability of another Event, and those two Events are independent ; the Probability that both those Events will Happen, will be the Product of those two Fractions.”(see also [Moivre, 1718](#), p. 4). Mathematically, in terms of probability theory, independence ([Kolmogoroff, Andreï Nikolaevich, 1933](#)) of events at the same (period of) time (i.e. Bernoulli trial)  $t$  is defined as

$$\begin{aligned} p(A_t \wedge B_t) &\equiv p(A_t) \times p(B_t) \equiv p(a_t) \\ &\equiv \frac{\sum_{t=1}^N (A_t \wedge B_t)}{N} \equiv \frac{N \times (p(a_t))}{N} \equiv 1 - p(A_t | B_t) \equiv 1 - p(A_t \uparrow B_t) \end{aligned} \quad (247)$$

while  $p(A_t \cap B_t)$  is the joint probability of the events  $A_t$  and  $B_t$  at a same Bernoulli trial  $t$ ,  $p(A_t)$  is the probability of an event  $A_t$  at a same Bernoulli trial  $t$ , and  $p(B_t)$  is the probability of an event  $B_t$  at a same Bernoulli trial  $t$ . With respect to a two-by-two table , **under conditions of independence**, it is

$$p(b_t) \equiv p(A_t) \times p(\underline{B}_t) \quad (248)$$

or

$$p(c_t) \equiv p(\underline{A}_t) \times p(B_t) \quad (249)$$

and

$$p(d_t) \equiv p(\underline{A}_t) \times p(\underline{B}_t) \quad (250)$$

**Example.** In a narrower sense, the *conditio sine qua non* relationship concerns itself at the end only with the case whether the presence of an event  $A_t$  (condition) enables or guarantees the presence of another event  $B_t$  (conditioned). Thus far, as a result of the thoughts before, another question worth asking concerns the relationship between the independence of an event  $A_t$  (a condition) and another event  $B_t$  (conditioned) and the necessary condition relationship. To be confronted with the danger of bias and equally with the burden of inappropriate conclusions drawn, another fundamental question at this stage is whether it is possible that an event  $A_t$  (a condition) is a necessary condition of event  $B_t$  (conditioned) even under circumstances where the event  $A_t$  (a condition) (a necessary condition) is independent of an event  $B_t$  (conditioned)? Meanwhile, this question is more or less already answered to the negative (Barukčić, 2018b). An event  $A_t$  which is a necessary condition of another event  $B_t$  is equally an event without which another event ( $B_t$ ) could not be, could not occur, and implies as such already a kind of dependence. However, it is not mandatory that such a kind of dependence is a causal one. It is remarkable that **data which provide evidence of a significant *conditio sine qua non* relationship between two events like  $A_t$  and  $B_t$  and equally support the hypothesis that  $A_t$  and  $B_t$  are independent of each other are more or less self-contradictory and of very restricted or of none value for further analysis.** In fact, if the opposite view would be taken as plausible, contradictions are more or less inescapable.

### 2.2.9. Dependence

#### Definition 2.15 (Dependence).

Whilst it may be true that the occurrence of an event  $A_t$  does not affect the occurrence of an other event  $B_t$  the contrary is of no minor importance. Under these other conditions, events, trials and random variables et cetera are dependent on each other too. The dependence of events (Barukčić, 1989, p. 57-61) is defined as

$$p\left(\underbrace{A_t \wedge B_t \wedge C_t \wedge \dots}_{n \text{ random variables}}\right) \equiv \sqrt[n]{\underbrace{p(A_t) \times p(B_t) \times p(C_t) \times \dots}_{n \text{ random variables}}} \quad (251)$$



## 2.2.10. Sensitivity and specificity

### Definition 2.16 (Sensitivity and specificity).

A (medical) test should measure what is supposed to measure. However, the extent to which a test measures what it is supposed to measure varies and is seldom equal to 100 %. In other words, it is necessary to check once and again the accuracy or the validity of a test, we have to fight it out in detail. In clinical practice, the concept of sensitivity and specificity is commonly used to quantify the diagnostic ability of a (medical) test. Sensitivity and specificity were introduced by the American<sup>97</sup>,<sup>98</sup>,<sup>99</sup>,<sup>100</sup> biostatistician Jacob Yerushalmy (see also Yerushalmy, 1947) in the year 1947. The interior logic of sensitivity and specificity is best illustrated using a conventional two- by-two (2 x 2) table (see table 5).

**Table 5.** Sensitivity and specificity

		Disease $B_t$		
		present	absent	
$A_t$	positive	a (true positive)	b (false positive)	A
	negative	c (false negative)	d (true negative)	$\underline{A}$
		<u>B</u>	<u><math>\underline{B}</math></u>	N

The ability of a positive test ( $A_t$ ) to correctly classify an individual as diseased ( $B_t$ ) is defined as the proportion of true positives that are correctly identified by the test (a) divided by the individuals being truly diseased ( $B_t$ ). In general, sensitivity follows as

$$\text{Sensitivity}(A | B) \equiv p(a | B) \equiv \frac{a}{B} \quad (252)$$

The specificity of a test is the ability of a negative test ( $\underline{A}_t$ ) to correctly classify an individual as not diseased ( $\underline{B}_t$ ) and is defined as the proportion of true negative that are correctly identified by the test (d) divided by the individuals being truly not diseased ( $\underline{B}_t$ ). In general, specificity is given by the equation

$$\text{Specificity}(\underline{A}, \underline{B}) \equiv p(d | \underline{B}) \equiv \frac{d}{\underline{B}} \quad (253)$$

The positive predictive value (PPV) is defined as

$$\text{PPV}(A, B) \equiv \frac{a}{a + b} \quad (254)$$

<sup>97</sup>Yerushalmy Jacob. Statistical problems in assessing methods of medical diagnosis, with special reference to X-ray techniques. Public Health Rep. 1947 Oct 3;62(40):1432-49. PMID: 20340527.

<sup>98</sup>Galen RS, Gambino SR. Beyond normality-the predictive value and efficiency of medical diagnosis. New York: NY:Wiley; 1975.

<sup>99</sup>Altman DG, Bland JM. Diagnostic tests. 1: Sensitivity and specificity. BMJ. 1994 Jun 11;308(6943):1552. doi: 10.1136/bmj.308.6943.1552. PMID: 8019315; PMCID: PMC2540489.

<sup>100</sup>Parikh R, Mathai A, Parikh S, Chandra Sekhar G, Thomas R. Understanding and using sensitivity, specificity and predictive values. Indian J Ophthalmol. 2008 Jan-Feb;56(1):45-50. doi: 10.4103/0301-4738.37595. PMID: 18158403; PMCID: PMC2636062.

The negative predictive value (NPV) is defined as

$$NPV(A, B) \equiv \frac{d}{c+d} \quad (255)$$

### Example.

The importance of sensitivity and specificity in any research should certainly not be underestimated. However, it is essential not to lose sight of the major advantages and limitations<sup>101</sup> of these measures. In the following, in order to avoid misconceptions about sensitivity, specificity et cetera, let us consider a test with a sensitivity of 95 % and a specificity of 95 %. A two-by-two table is used as an illustration (see table 6).

**Table 6.** Sensitivity and specificity

		Disease B <sub>t</sub>		
		present	absent	
Test A <sub>t</sub>	positive	95	5	100
	negative	5	95	100
		100	100	200

Sensitivity is calculated as

$$Sensitivity(A | B) \equiv p(a | B) \equiv 100 \times \frac{a}{B} \equiv \frac{95}{100} \equiv 95\% \quad (256)$$

There are at least two kinds of medical tests, diagnostic tests and screening tests. Depending on the type of medical test, there are other logical implications. A screening test should correctly identify all people who suffer from a certain disease or all people with a certain outcome. Therefore, the sensitivity of a screening test should be at best 100 %. Under these conditions, we obtain **without** positive test **no** disease/outcome present. However, confusion should be avoided with regard to the adequacy and usefulness of the sensitivity of a screening test. The sensitivity of a test does not take into account events which are false positive (b) or which are true negative (d), the meaning of these events is ignored completely by sensitivity. Therefore, sensitivity is blind on one eye since its inception and underestimates the extent to which a screening test is able to identify the likely presence of a condition of interest. We calculated a 95 % sensitivity while the true possibility of the test to detect a disease is (see table 6)

$$SINE(A, B) \equiv 100 \times \frac{a+b+d}{N} \equiv \frac{95+5+95}{200} \equiv 97.5\% \quad (257)$$

In a way similar to sensitivity, specificity is not much better. Diagnostic tests are able to identify people who do not have a certain condition. Specificity is calculated as

$$Specificity(\underline{A} | \underline{B}) \equiv p(d | \underline{B}) \equiv 100 \times \frac{d}{\underline{B}} \equiv \frac{95}{100} \equiv 95\% \quad (258)$$

<sup>101</sup>Trevethan R. Sensitivity, Specificity, and Predictive Values: Foundations, Plabilities, and Pitfalls in Research and Practice. *Front Public Health*. 2017 Nov 20;5:307. doi: 10.3389/fpubh.2017.00307. PMID: 29209603; PMCID: PMC5701930.

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However, specificity does not take into account any individuals who suffer from a disease, who do have the condition and is well-known for being imperfect because of this fact too. Specificity underestimates the possibility of a diagnostic test to detect a disease. Above, the specificity has been calculated as being 95 %. In point of fact, the ability of the test to detect a disease or the relationship **if** test positive **then** disease present is much better and has to be calculated as (see table 6)

$$IMP(A, B) \equiv \frac{a + c + d}{N} \equiv \frac{95 + 5 + 95}{200} \equiv 97.5\% \quad (259)$$

As can be seen, the test detected the disease in 97.5 % while specificity allows only 95 %. How valuable is such a measure epistemologically? Measures like sensitivity and specificity are blurring of the issue, do risk leading us astray and disorient us systematically again and again. These measures should be abandoned.

## 2.2.11. Odds ratio (OR)

**Definition 2.17** (Odds ratio (OR)).

Odds ratios as an appropriate measure for estimating the relative risk have become widely used in medical reports of case-control studies. The odds ratio (Fisher, 1935, p. 50) is defined (Cox, 1958) as the ratio of the odds of an event occurring in one group with respect to the odds of its occurring in another group. Odds (Yule and Pearson, 1900, p. 273) ratio (OR) is a measure of association which quantifies the relationship between two binomial distributed random variables (exposure vs. outcome) and is related to Yule's (Yule and Pearson, 1900, p. 272)  $Q$  (Yule, 1912, p. 585/586). Two events  $A_t$  and  $B_t$  are regarded as independent if  $(A_t, B_t) = 1$ . Let

$a_t$  = number of persons exposed to  $A_t$  and with disease  $B_t$

$b_t$  = number of persons exposed to  $A_t$  but without disease  $B_t$

$c_t$  = number of persons unexposed  $\bar{A}_t$  but with disease  $B_t$

$d_t$  = number of persons unexposed  $\bar{A}_t$ : and without disease  $B_t$

$a_t + c_t$  = total number of persons with disease  $B_t$  (case-patients)

$b_t + d_t$  = total number of persons without disease  $B_t$  (controls).

Hereafter, consider the table 7. The odds' ratio (OR) is defined as

**Table 7.** The two by two table of random variables

		Conditioned/Outcome $B_t$		
		TRUE	FALSE	
Condition/Exposure $A_t$	TRUE	$a_t$	$b_t$	$A_t$
	FALSE	$c_t$	$d_t$	$\bar{A}_t$
		$B_t$	$\bar{B}_t$	$N_t$

$$\begin{aligned}
 OR(A_t, B_t) &\equiv \left( \frac{a_t}{b_t} \right) / \left( \frac{c_t}{d_t} \right) \\
 &\equiv \left( \frac{a_t \times d_t}{b_t \times c_t} \right)
 \end{aligned} \tag{260}$$

**Remark 2.2.** Odds ratios can support logical fallacies and cause difficulties in drawing logically consistent conclusions. The chorus of voices is growing, which demand the immediate ending (Knol, 2012, Sackett, DL and Deeks, JJ and Altman, DG, 1996) of any use of Odds ratio.

Under conditions where  $(b = 0)$ , the measure of association odds ratio will collapse, because we need to divide by zero, as can be seen at eq. 260. However, according to today's rules of mathematics,

---

*a division by zero is neither allowed nor generally accepted as possible. It does no harm to remind ourselves that in the case  $b = 0$  the event  $A_t$  is a sufficient condition of  $B_t$ . In other words, odds ratio is not able to recognize elementary relationships of objective reality. In fact, it would be a failure not to recognize how dangerous and less valuable odds ratio is.*

*Under conditions where  $(c = 0)$  odds ratio collapses too, because we need again to divide by zero, as can be seen at eq. 260. However, and again, today's rules of mathematics don't allow us a division by zero. In point of fact, in the case  $c = 0$  it is more than necessary to point out that  $A_t$  is a necessary condition of  $B_t$ . In other words, odds ratio or the cross-product ratio is not able to recognize elementary relationships of nature like necessary conditions. We can and need to overcome all the epistemological obstacles as backed by odds ratio entirety. Sooner rather than later, we should give up this measure of relationship completely.*

## 2.2.12. Relative risk (RR)

### 2.2.12.1. Relative risk ( $RR_{nc}$ )

**Definition 2.18** (Relative risk ( $RR_{nc}$ )).

The degree of association between the two binomial variables can be assessed by a number of very different coefficients, the relative (Cornfield, 1951, Sadowsky et al., 1953) risk is one (Barukčić, 2021d) of them. In general, relative risk  $RR_{nc}$ , which provides some evidence of a necessary condition, is defined as

$$\begin{aligned}
 RR(A_t, B_t)_{nc} &\equiv \frac{\frac{p(a_t)}{p(A_t)}}{\frac{p(c_t)}{p(NotA_t)}} \\
 &\equiv \frac{p(a_t) \times p(NotA_t)}{p(c_t) \times p(A_t)} \\
 &\equiv \frac{N \times p(a_t) \times N \times p(NotA_t)}{N \times p(c_t) \times N \times p(A_t)} \\
 &\equiv \frac{a_t \times (NotA_t)}{c_t \times A_t} \\
 &\equiv \frac{EER(A_t, B_t)}{CER(A_t, B_t)}
 \end{aligned} \tag{261}$$

That what scientist generally understand by relative risk is the ratio of a probability of an event occurring with an exposure versus the probability of an event occurring without an exposure. In other words,

**relative risk = (probability(event in exposed group)) / (probability(the same event in not exposed group)).**

A  $RR(A_t, B_t) = +1$  means that exposure does not affect the outcome or both are independent of each other while  $RR(A_t, B_t)$  less than +1 means that the risk of the outcome is decreased by the exposure. In this context, an  $RR(A_t, B_t)$  greater than +1 denotes that the risk of the outcome is increased by the exposure. Widely known problems with odds ratio and relative risk are already documented in literature.

### 2.2.12.2. Relative risk (RR (sc))

**Definition 2.19** (Relative risk (RR (sc))).

The relative risk (sc), which provides some evidence of a sufficient condition, is calculated from the point of view of an outcome and is defined as

$$\begin{aligned}
 RR(A_t, B_t)_{sc} &\equiv \frac{\frac{p(a_t)}{p(B_t)}}{\frac{p(b_t)}{p(NotB_t)}} \\
 &\equiv \frac{p(a_t) \times p(NotB_t)}{p(b_t) \times p(B_t)} \\
 &\equiv \frac{N \times p(a_t) \times N \times p(NotB_t)}{N \times p(b_t) \times N \times p(B_t)} \\
 &\equiv \frac{a_t \times (NotB_t)}{b_t \times B_t} \\
 &\equiv \frac{OPR(A_t, B_t)}{CPR(A_t, B_t)}
 \end{aligned} \tag{262}$$

### 2.2.12.3. Relative risk reduction (RRR)

**Definition 2.20** (Relative risk reduction (RRR)).

$$\begin{aligned}
 RRR(A_t, B_t) &\equiv \frac{CER(A_t, B_t) - EER(A_t, B_t)}{CER(A_t, B_t)} \\
 &= 1 - RR(A_t, B_t)
 \end{aligned} \tag{263}$$

### 2.2.12.4. Vaccine efficacy (VE)

**Definition 2.21** (Vaccine efficacy (VE)).

Vaccine efficacy is defined as the percentage reduction of a disease in a vaccinated group of people as compared to an unvaccinated group of people.

$$\begin{aligned}
 VE(A_t, B_t) &\equiv 100 \times (1 - RR(A_t, B_t)) \\
 &\equiv 100 \times \left( \frac{CER(A_t, B_t) - EER(A_t, B_t)}{CER(A_t, B_t)} \right)
 \end{aligned} \tag{264}$$

Historically, vaccine efficacy has been designed to evaluate the efficacy of a certain vaccine by Greenwood and Yule in 1915 for the cholera and typhoid vaccines (Greenwood and Yule, 1915) and best measured using double-blind, randomized, clinical controlled trials. However, the calculated vaccine efficacy is depending too much on the study design, can lead to erroneous conclusions and is only of very limited value.

#### 2.2.12.5. Experimental event rate (EER)

**Definition 2.22** (Experimental event rate (EER)).

$$EER(A_t, B_t) \equiv \frac{p(a_t)}{p(A_t)} = \frac{a_t}{a_t + b_t} \quad (265)$$

**Definition 2.23** (Control event rate (CER)).

$$CER(A_t, B_t) \equiv \frac{p(c_t)}{p(\underline{A}_t)} = \frac{c_t}{c_t + d_t} \quad (266)$$

#### 2.2.12.6. Absolute risk reduction (ARR)

**Definition 2.24** (Absolute risk reduction (ARR)).

$$\begin{aligned} ARR(A_t, B_t) &\equiv \frac{p(c_t)}{p(\underline{A}_t)} - \frac{p(a_t)}{p(A_t)} \\ &= \frac{c_t}{c_t + d_t} - \frac{a_t}{a_t + b_t} \\ &= CER(A_t, B_t) - EER(A_t, B_t) \end{aligned} \quad (267)$$

#### 2.2.12.7. Absolute risk increase (ARI)

**Definition 2.25** (Absolute risk increase (ARI)).

$$\begin{aligned} ARI(A_t, B_t) &\equiv \frac{p(a_t)}{p(A_t)} - \frac{p(c_t)}{p(\underline{A}_t)} \\ &= EER(A_t, B_t) - CER(A_t, B_t) \end{aligned} \quad (268)$$



### 2.2.12.8. Number needed to treat (NNT)

**Definition 2.26** (Number needed to treat (NNT)).

$$NNT(A_t, B_t) \equiv \frac{1}{CER(A_t, B_t) - EER(A_t, B_t)} \quad (269)$$

An ideal number needed to treat (Cook and Sackett, 1995, Laupacis et al., 1988), mathematically the reciprocal of the absolute risk reduction, is  $NNT = 1$ . Under these circumstances, everyone improves with a treatment, while no one improves with control. A higher number needed to treat indicates more or less a treatment which is less effective.

### 2.2.12.9. Number needed to harm (NNH)

**Definition 2.27** (Number needed to harm (NNH)).

$$NNH(A_t, B_t) \equiv \frac{1}{EER(A_t, B_t) - CER(A_t, B_t)} \quad (270)$$

The number needed to harm (Massel and Cruickshank, 2002), mathematically the inverse of the absolute risk increase, indicates at the end how many patients need to be exposed to a certain factor, in order to observe a harm in one patient that would not otherwise have been harmed.

### 2.2.12.10. Outcome prevalence rate (OPR)

**Definition 2.28** (Outcome prevalence rate (OPR)).

$$OPR(A_t, B_t) \equiv \frac{p(a_t)}{p(B_t)} = \frac{a_t}{a_t + c_t} \quad (271)$$

### 2.2.12.11. Control prevalence rate (CPR)

**Definition 2.29** (Control prevalence rate (CPR)).

$$CPR(A_t, B_t) \equiv \frac{p(b_t)}{p(B_t)} = \frac{b_t}{b_t + d_t} \quad (272)$$

Bias and confounding is present to some degree in all research. In order to assess the relationship of exposure with a disease or an outcome, a fictive control group (i.e. of newborn or of young children et cetera) can be of use too. Under certain circumstances, even a  $CPR = 0$  is imaginable.

### 2.2.12.12. Absolute prevalence reduction (APR)

**Definition 2.30** (Absolute prevalence reduction (APR)).

$$APR(A_t, B_t) \equiv CPR(A_t, B_t) - OPR(A_t, B_t) \quad (273)$$

### 2.2.12.13. Absolute prevalence increase (API)

**Definition 2.31** (Absolute prevalence increase (API)).

$$API(A_t, B_t) \equiv OPR(A_t, B_t) - CPR(A_t, B_t) \quad (274)$$

### 2.2.12.14. Relative prevalence reduction (RPR)

**Definition 2.32** (Relative prevalence reduction (RPR)).

$$\begin{aligned} RPR(A_t, B_t) &\equiv \frac{CPR(A_t, B_t) - OPR(A_t, B_t)}{CPR(A_t, B_t)} \\ &= 1 - RR(A_t, B_t)_{sc} \end{aligned} \quad (275)$$

### 2.2.12.15. The index NNS

**Definition 2.33** (The index NNS).

$$NNS(A_t, B_t) \equiv \frac{1}{CPR(A_t, B_t) - OPR(A_t, B_t)} \quad (276)$$

Mathematically, the index NNS is the reciprocal of the absolute prevalence reduction.

### 2.2.12.16. The index NNI

**Definition 2.34** (The index NNI).

$$NNI(A_t, B_t) \equiv \frac{1}{OPR(A_t, B_t) - CPR(A_t, B_t)} \quad (277)$$

Mathematically, the index NNI is the reciprocal of the absolute prevalence increase.

### 2.2.13. Index of relationship (IOR)

**Definition 2.35** (Index of relationship (IOR)).

Due to several reasons, it is not always easy to identify the unique characteristics between two events like  $A_t$  and  $B_t$ . And more than that, it is difficult to decide what to do, and much more difficult to know in which direction one should think and which decision is right. Sometimes it is helpful to know at least something about the direction of the relationship between two events like  $A_t$  and  $B_t$ . Under conditions where  $p(a_t) = p(A_t \wedge B_t)$ , the index of relationship (Barukčić, 2021b), abbreviated as IOR, is defined as

$$\begin{aligned}
 IOR(A_t, B_t) &\equiv \left( \frac{p(A_t \wedge B_t)}{p(B_t) \times p(A_t)} \right) - 1 \\
 &\equiv \left( \frac{p(a_t)}{p(B_t) \times p(A_t)} \right) - 1 \\
 &\equiv \left( \left( \frac{N \times N \times p(a_t)}{N \times p(B_t) \times N \times p(A_t)} \right) - 1 \right) \\
 &\equiv \left( \left( \frac{N \times a}{A \times B} \right) - 1 \right)
 \end{aligned} \tag{278}$$

where  $p(A_t)$  denotes the probability of an event  $A_t$  at the Bernoulli trial  $t$  and  $p(B_t)$  denotes the probability of another event  $B_t$  at the same Bernoulli trial  $t$  while  $p(a_t)$  denotes the joint probability of  $p(A_t \text{ AND } B_t)$  at the same Bernoulli trial  $t$  and  $a$ ,  $A$  and  $B$  may denote the expectation values.

**Definition 2.36** (Multi dimensional index of relationship (NIOR)).

The multi dimensional index of relationship (NIOR) is defined as

$$\begin{aligned}
 NIOR(A_t, B_t) &\equiv \left( \frac{N^k \times p({}_1A_t \wedge {}_2A_t \cdots {}_kA_t)}{N \times (p({}_1A_t)) N \times (p({}_2A_t)) \cdots N \times (p({}_kA_t))} \right) - 1 \\
 &\equiv \left( \frac{N^{k-1} \times E({}_1A_t \wedge {}_2A_t \cdots {}_kA_t)}{E({}_1A_t) \times E({}_2A_t) \cdots \times E({}_kA_t)} \right) - 1
 \end{aligned} \tag{279}$$

where  $N$  is the sample size and  $p({}_1A_t \wedge {}_2A_t \cdots {}_kA_t)$  is the joint distribution function.

However, there might exist circumstances where a multi dimensional index of relationship might take the form of the following equation.

$$\begin{aligned}
 NIOR(A_t, B_t) &\equiv \left( \frac{{}_1N \times {}_2N \times \cdots \times {}_kN \times p({}_1A_t \wedge {}_2A_t \cdots {}_kA_t)}{({}_1N \times p({}_1A_t)) \times ({}_2N \times p({}_2A_t)) \cdots \times ({}_kN \times p({}_kA_t))} \right) - 1 \\
 &\equiv \left( \frac{{}_1N \times {}_2N \times \cdots \times {}_kN \times p({}_1A_t \wedge {}_2A_t \cdots {}_kA_t)}{E({}_1A_t) \times E({}_2A_t) \cdots \times E({}_kA_t)} \right) - 1
 \end{aligned} \tag{280}$$

### 2.3. Conditions

Even if a condition and a cause are deeply related, there are circumstances where a sharp distinction between a cause and a condition is necessary. However, exactly this has been denied by John Stuart Mill's (1806-1873) regularity view of causality (see [Mill, 1843b](#)). What might seem to be a theoretical difficulty for many authors is none for Mill. Mill simply reduced a cause to a condition and claimed that "... the real cause of the phenomenon is the assemblage of all its conditions." (see [Mill, 1843a](#), p. 403)

#### 2.3.1. Exclusion relationship

**Definition 2.37 (Exclusion relationship [EXCL]).**

Mathematically, the exclusion(see also [Barukčić, 2021a](#)) relationship <sup>102</sup> (EXCL), denoted by  $p(A_t | B_t)$  in terms of statistics and probability theory, is defined(see also [Barukčić, 1989](#), p. 68-70) as

$$\begin{aligned}
 p(A_t | B_t) &\equiv p(A_t \uparrow B_t) \\
 &\equiv p(b_t) + p(c_t) + p(d_t) \\
 &\equiv \frac{N \times (p(b_t) + p(c_t) + p(d_t))}{N} \\
 &\equiv \frac{\sum_{t=1}^N (\underline{A}_t \vee \underline{B}_t)}{N} \equiv \frac{b + c + d}{N} \\
 &\equiv \frac{b + \underline{A}}{N} \\
 &\equiv \frac{c + \underline{B}}{N} \\
 &\equiv +1
 \end{aligned} \tag{281}$$

Based on the 1913 Henry Maurice Sheffer (1882-1964) relationship, the Sheffer stroke([Nicod, 1917](#), [Sheffer, 1913](#)) usually denoted by  $\uparrow$ , it is  $p(A_t \wedge B_t) \equiv 1 - p(A_t | B_t)$  (see table 8).

**Table 8.**  $A_t$  excludes  $B_t$  and vice versa.

		Conditioned (COVID-19) $B_t$		
		TRUE	FALSE	
Condition (Vaccine) $A_t$	TRUE	<b>+0</b>	$p(b_t)$	$p(\underline{A}_t)$
	FALSE	$p(c_t)$	$p(d_t)$	$p(\underline{A}_t)$
		$p(\underline{B}_t)$	$p(\underline{B}_t)$	+1

<sup>102</sup>Barukčić, Ilija. (2021). Mutually exclusive events. *Causation*, 16(11), 5–57. <https://doi.org/10.5281/zenodo.5746415>

**Example 2.1.** Pfizer Inc. and BioNTech SE announced on Monday, November 09, 2020 - 06:45am results from a Phase 3 COVID-19 vaccine trial with 43.538 participants which provides evidence that their vaccine (BNT162b2) is preventing COVID-19 in participants without evidence of prior SARS-CoV-2 infection. In toto, 170 confirmed cases of COVID-19 were evaluated, with 8 in the vaccine group versus 162 in the placebo group. The exclusion relationship can be calculated as follows.

$$\begin{aligned}
 p(\text{Vaccine : BNT162b2} \mid \text{COVID} - 19(\text{infection})) &\equiv p(b_t) + p(c_t) + p(d_t) \\
 &\equiv 1 - p(a_t) \\
 &\equiv 1 - \left( \frac{8}{43538} \right) \\
 &\equiv +0,99981625
 \end{aligned} \tag{282}$$

with a P Value = 0,000184.

Following Kolmogorov's definition of an n-dimensional probability density (see also [Kolmogorov, Andreï Nikolaevich, 1950](#), p. 26) of random variables  $A_t$ ,  $B_t$  et cetera at the point  $t$ , we obtain

$$\begin{aligned}
 p(A_t \mid B_t) &\equiv p(\underline{A}_t \cup \underline{B}_t) \\
 &\equiv 1 - p(A_t \cap B_t) \\
 &\equiv 1 - \int_{-\infty}^{A_t} \int_{-\infty}^{B_t} f(A_t, B_t) dA_t dB_t \\
 &\equiv +1
 \end{aligned} \tag{283}$$

while  $p(A_t \mid B_t)$  would denote the cumulative distribution function of random variables and  $f(A_t, B_t)$  is the joint density function.

### 2.3.2. Observational study and exclusion relationship

Under conditions of an observational study, the exclusion relationship follows approximately (see [Barukčić, 2021a](#)) as

$$p(A_t \mid B_t) \equiv p(A_t \uparrow B_t) \geq 1 - \frac{p(a_t)}{p(B_t)} \tag{284}$$

### 2.3.3. Experimental study and exclusion relationship

Under conditions of an experimental study, the exclusion relationship follows approximately (see [Barukčić, 2021a](#)) as

$$p(A_t \mid B_t) \equiv p(A_t \uparrow B_t) \geq 1 - \frac{p(a_t)}{p(A_t)} \tag{285}$$

### 2.3.4. The goodness of fit test of an exclusion relationship

#### Definition 2.38 (The $\tilde{\chi}^2$ goodness of fit test of an exclusion relationship).

Under some well known circumstances, testing hypothesis about an exclusion relationship  $p(A_t | B_t)$  is possible by the chi-square distribution (also chi-squared or  $\tilde{\chi}^2$ -distribution) too. The  $\tilde{\chi}^2$  goodness of fit test of an exclusion relationship with degree of freedom (d. f.) of d. f. = 1 is calculated as

$$\begin{aligned}\tilde{\chi}^2_{\text{Calculated}}((A_t | B_t) | A) &\equiv \frac{(b - (a + b))^2}{A} + \frac{((c + d) - \underline{A})^2}{\underline{A}} \\ &\equiv \frac{a^2}{A} + 0 \\ &\equiv \frac{a^2}{A}\end{aligned}\tag{286}$$

or equally as

$$\begin{aligned}\tilde{\chi}^2_{\text{Calculated}}((A_t | B_t) | B) &\equiv \frac{(c - (a + c))^2}{B} + \frac{((b + d) - \underline{B})^2}{\underline{B}} \\ &\equiv \frac{a^2}{B} + 0 \\ &\equiv \frac{a^2}{B}\end{aligned}\tag{287}$$

and can be compared with a theoretical chi-square value at a certain level of significance  $\alpha$ . The  $\tilde{\chi}^2$ -distribution equals zero when the observed values are equal to the expected/theoretical values of an exclusion relationship/distribution  $p(A_t | B_t)$ , in which case the null hypothesis has to be accepted. Yate's (Yates, 1934) continuity correction was not used under these circumstances.

### 2.3.5. The left-tailed p Value of an exclusion relationship

#### Definition 2.39 (The left-tailed p Value of an exclusion relationship).

It is known that as a sample size, N, increases, a sampling distribution of a special test statistic approaches the normal distribution (central limit theorem). Under these circumstances, the left-tailed

(It) p Value (Barukčić, 2019e) of an exclusion relationship can be calculated as follows.

$$\begin{aligned} pValue_{It}(A_t | B_t) &\equiv 1 - e^{-(1-p(A_t|B_t))} \\ &\equiv 1 - e^{-(a/N)} \end{aligned} \quad (288)$$

A low p-value may provide some evidence of statistical significance.

### 2.3.6. Neither nor conditions

#### Definition 2.40 (Neither $A_t$ nor $B_t$ conditions [NOR]).

Mathematically, a neither  $A_t$  nor  $B_t$  condition (or rejection according to the French philosopher and logician Jean George Pierre Nicod (1893-1924), i.e. Jean Nicod's statement (Nicod, 1924)) relationship (NOR), denoted by  $p(A_t \downarrow B_t)$  in terms of statistics and probability theory, is defined (Barukčić, 1989, p. 68-70) as

$$\begin{aligned} p(A_t \downarrow B_t) &\equiv p(d_t) \\ &\equiv \frac{N - \sum_{t=1}^N (A_t \vee B_t)}{N} \equiv \frac{\sum_{t=1}^N (\underline{A}_t \wedge \underline{B}_t)}{N} \equiv \frac{N \times (p(d_t))}{N} \\ &\equiv \frac{d}{N} \\ &\equiv +1 \end{aligned} \quad (289)$$

### 2.3.7. The Chi square goodness of fit test of a neither nor condition relationship

#### Definition 2.41 (The $\tilde{\chi}^2$ goodness of fit test of a neither $A_t$ nor $B_t$ condition relationship).

A neither  $A_t$  nor  $B_t$  condition relationship  $p(A_t \downarrow B_t)$  can be tested by the chi-square distribution (also chi-squared or  $\tilde{\chi}^2$ -distribution). The  $\tilde{\chi}^2$  goodness of fit test of a neither  $A_t$  nor  $B_t$  condition relationship with degree of freedom (d. f.) of d. f. = 1 may be calculated as

$$\begin{aligned} \tilde{\chi}^2_{\text{Calculated}}((A_t \downarrow B_t) | A) &\equiv \frac{(d - (c + d))^2}{\underline{A}} + \\ &\quad \frac{((a + b) - A)^2}{A} \\ &\equiv \frac{c^2}{\underline{A}} + 0 \end{aligned} \quad (290)$$

or equally as

$$\begin{aligned}\tilde{\chi}^2_{\text{Calculated}}((A_t \downarrow B_t) | B) &\equiv \frac{(d - (b + d))^2}{B} + \\ &\quad \frac{((a + c) - B)^2}{B} \\ &\equiv \frac{b^2}{B} + 0\end{aligned}\tag{291}$$

Yate's (Yates, 1934) continuity correction has not been used in this context.

### 2.3.8. The left-tailed p Value of a neither nor B condition relationship

**Definition 2.42 (The left-tailed p Value of a neither  $A_t$  nor  $B_t$  condition relationship).**

The left-tailed (lt) p Value (Barukčić, 2019e) of a neither  $A_t$  nor  $B_t$  condition relationship can be calculated as follows.

$$\begin{aligned}pValue_{lt}(A_t \downarrow B_t) &\equiv 1 - e^{-(1-p(A_t \downarrow B_t))} \\ &\equiv 1 - e^{-p(A_t \vee B_t)} \\ &\equiv 1 - e^{-((a+b+c)/N)}\end{aligned}\tag{292}$$

where  $\vee$  may denote disjunction or logical inclusive or. In this context, a low p-value indicates again a statistical significance. In general, it is  $p(A_t \vee B_t) \equiv 1 - p(A_t \downarrow B_t)$  (see table 9).

**Table 9.** Neither  $A_t$  nor  $B_t$  relationship.

		Conditioned $B_t$		
		YES	NO	
Condition $A_t$	YES	0	0	0
	NO	0	1	1
		0	1	1



### 2.3.9. Necessary condition

#### **Definition 2.43 (Necessary condition [*Conditio sine qua non*]).**

Despite the most extended efforts, the current state of research on conditions and conditioned is still incomplete and very contradictory. However, even thousands of years ago and independently of any human mind and consciousness, water has been and is still a necessary (see Barukčić, 2022b) condition for (human) life. **Without** water, there has been and there is **no** (human) life<sup>103</sup>. It comes therefore as no surprise that one of the first documented attempts to present a rigorous theory of conditions and causation (see also Aristotle, of Stageira (384-322 B.C.E), 1908, *Metaphysica* III 2 997a 10 and 13/14) came from the Greek philosopher and scientist Aristotle (384-322 BCE). Thus far, it is amazing that Aristotle himself made already a strict distinction between conditions and causes. Taking Aristotle very seriously, it is necessary to consider that

“... everything which has a ... .. potency in question ... .. has the potency ... of acting ... not in all circumstances but on certain conditions ... ” (see also Aristotle, of Stageira (384-322 B.C.E), 1908, *Metaphysica* IX 5 1048a 14-19)

Before going into details, Aristotle went on to define the necessary condition as follows.

“... necessary ... means ...

without ... a condition, a thing cannot live ... ”

(see also Aristotle, of Stageira (384-322 B.C.E), 1908, *Metaphysica* V 2 1015a 20-22)

In point of fact, Aristotle developed a theory of conditions and causality commonly referred to as the doctrine of four causes. Many aspects and general features of Aristotle’s logical concept of causality are meanwhile extensively and critically debated in secondary literature. However, even if the Greek philosophers Heraclitus, Plato, Aristotle et cetera numbers among the greatest philosophers of all time, the philosophy has evolved. Scientific knowledge and objective reality are deeply interrelated and cannot be reduced only to Greek philosophers like Aristotle. Among many other issues, the specification of necessary conditions has traditionally been part of the philosopher’s investigations of different phenomena. However, behind the need of a detailed evidence, it is justified to consider that philosophy or philosophers as such certainly do not possess **a monopoly on the truth** and other areas such as medicine as well as other sciences and technology may transmit truths as well and may be of help to move beyond one’s self enclosed unit. Seemingly, **the law’s concept of causation** justifies to say few words on this subject, to put some light on some questions. Are there any criteria in law for deciding whether one action or an event  $A_t$  has caused another (generally harmful) event  $B_t$ ? What are these criteria? May causation in legal contexts differ from causation outside the law, for example, in science

<sup>103</sup>Barukčić, Ilija. (2022). *Conditio sine qua non* (Version 1). Zenodo. <https://doi.org/10.5281/zenodo.5854744>

or in our everyday life and to what extent? Under which circumstances is it justified to tolerate such differences as may be found to exist? To understand just what is the law's concept of causation, it is useful to re-consider how the highest court of states is dealing with causation. In the case *Hayes v. Michigan Central R. Co.*, 111 U.S. 228, the U.S. Supreme Court defined 1884 *conditio sine qua non* as follows: "... **causa sine qua non – a cause which, if it had not existed, the injury would not have taken place**". (Justice Matthews, Mr., 1884) The German Bundesgerichtshof für Strafsachen stressed once again the importance of *conditio sine qua non* relationship in his decision by defining the following: "**Ursache eines strafrechtlich bedeutsamen Erfolges jede Bedingung, die nicht hinweggedacht werden kann, ohne daß der Erfolg entfiel**"(Bundesgerichtshof für Strafsachen, 1951) Another lawyer elaborated on the basic issue of **identity and difference between cause and condition**. Von Bar was writing: "Die erste Voraussetzung, welche erforderlich ist, damit eine Erscheinung als die Ursache einer anderen bezeichnet werden könne, ist, daß jene eine der Bedingungen dieser sein. Würde die zweite Erscheinung auch dann eingetreten sein, wenn die erste nicht vorhanden war, so ist sie in keinem Falle Bedingung und noch weniger Ursache. Wo immer ein Kausalzusammenhang behauptet wird, da muß er wenigstens diese Probe aushalten ... **Jede Ursache ist notwendig auch eine Bedingung eines Ereignisses; aber nicht jede Bedingung ist Ursache zu nennen.**"(Bar, Carl Ludwig von, 1871) Von Bar's position translated into English: *The first requirement, which is required, thus that something could be called as the cause of another, is that the one has to be one of the conditions of the other. If the second something had occurred even if the first one did not exist, so it is by no means a condition and still less a cause. Wherever a causal relationship is claimed, the same must at least withstand this test. . . . Every cause is necessarily also a condition of an event too; but not every condition is cause too.* Thus far, let us consider among other the following in order to specify necessary conditions from another, probabilistic point of view. An event (i.e.  $A_t$ ) which is a necessary condition of another event or outcome (i.e.  $B_t$ ) must be given, must be present for a conditioned, for an event or for an outcome  $B_t$  to occur. A necessary condition (i.e.  $A_t$ ) is a requirement which need to be fulfilled **at every single Bernoulli trial t**, in order for a conditioned or an outcome (i.e.  $B_t$ ) to occur, but it alone does not determine the occurrence of such an event. In other words, if a necessary condition (i.e.  $A_t$ ) is given, an outcome (i.e.  $B_t$ ) need not to occur. In contrast to a necessary condition, a 'sufficient' condition is the one condition which 'guarantees' that an outcome will take place or will occur for sure. Under which conditions we may infer about the unobserved and whether observations made are able at all to justify predictions about potential observations which have not yet been made or even general claims which may go even beyond the observed (*the 'problem of induction'*) is not the issue of the discussion at this point. Besides of the principal necessity of meeting such a challenge, a necessary condition of an event can but need not be at the same Bernoulli trial t a sufficient condition for an event to occur. However, theoretically, it is possible that an event or an outcome is determined by many necessary conditions. Let us focus to some extent on what this means, or in other words how much importance can we attribute to such a special case. *Example.* A human being cannot live without oxygen. A human being cannot live without water. A human being cannot live without a brain. A human being cannot live without kidneys. A human being cannot live without ... et cetera. Thus far, even if oxygen is given, if a brain is given ... et cetera, without water a human being will not survive on the long run. This example is of use to reach the following conclusion. Although it might seem somewhat paradoxical at first sight, **even under circumstances where a condition or an outcome depends on several different necessary conditions it is particularly important that every single of**

these necessary conditions for itself must be given otherwise the conditioned (i.e. the outcome) will not occur. Mathematically, the necessary condition (SINE) relationship, denoted by  $p(A_t \leftarrow B_t)$  in terms of statistics and probability theory, is defined (Barukčić, 1989, p. 15-28) as

$$\begin{aligned}
 p(A_t \leftarrow B_t) &\equiv p(A_t \vee \underline{B}_t) \equiv \frac{\sum_{t=1}^N (A_t \vee \underline{B}_t)}{N} \equiv \frac{(A_t \vee \underline{B}_t) \times p(A_t \vee \underline{B}_t)}{(A_t \vee \underline{B}_t)} \\
 &\equiv p(a_t) + p(b_t) + p(d_t) \\
 &\equiv \frac{N \times (p(a_t) + p(b_t) + p(d_t))}{N} \equiv \frac{E(A_t \leftarrow B_t)}{N} \\
 &\equiv \frac{a + b + d}{N} \equiv \frac{E(A_t \vee \underline{B}_t)}{N} \\
 &\equiv \frac{A + d}{N} \equiv \frac{E(A_t \leftarrow B_t)}{N} \\
 &\equiv \frac{a + \underline{B}}{N} \equiv \frac{E(A_t \vee \underline{B}_t)}{N} \\
 &\equiv +1
 \end{aligned} \tag{293}$$

where  $E(A_t \leftarrow B_t) \equiv E(A_t \vee \underline{B}_t)$  indicates the expectation value of the necessary condition. In general, it is  $p(A_t \leftarrow B_t) \equiv 1 - p(A_t \leftarrow B_t)$  (see Table 10).

**Table 10.** Necessary condition.

		Conditioned $B_t$		
		TRUE	FALSE	
Condition	TRUE	$p(a_t)$	$p(b_t)$	$p(A_t)$
	FALSE	<b>+0</b>	$p(d_t)$	$p(\underline{A}_t)$
		$p(B_t)$	$p(\underline{B}_t)$	+1

A necessary condition  $A_t$  is characterised itself by the property that another event  $B_t$  will not occur if  $A_t$  is not given, if  $A_t$  did not occur (Barukčić, 1989, 1997, 2005, 2016b, 2017b,c, 2020a,b,c,d, Barukčić and Ufuoma, 2020). Taking into account Kolmogorov's definition of an n-dimensional probability density (see also Kolmogorov, Andreĭ Nikolaevich, 1950, p. 26) of random variables  $A_t, B_t$  et cetera at the (period of) time  $t$ , we obtain

$$\begin{aligned}
 p(A_t \leftarrow B_t) &\equiv +1 \\
 &\equiv +1 - p(c_t) \\
 &\equiv +1 - p(\underline{A}_t \cap B_t) \\
 &\equiv \left( \int_{-\infty}^{A_t} \int_{-\infty}^{B_t} f(A_t, B_t) dA_t dB_t \right) + \left( 1 - \int_{-\infty}^{B_t} f(B_t) dB_t \right)
 \end{aligned} \tag{294}$$

while  $p(A_t \leftarrow B_t)$  would denote the cumulative distribution function of random variables of a necessary condition. Another adequate formulation of a necessary condition is possible too. If certain conditions

are met, then necessary conditions and sufficient conditions are one way or another converses of each other, too. It is

$$p(A_t \leftarrow B_t) \equiv \underbrace{(A_t \vee B_t)}_{\text{(Necessary condition)}} \equiv \underbrace{(B_t \vee A_t)}_{\text{(Sufficient condition)}} \equiv p(B_t \rightarrow A_t) \quad (295)$$

These relationships are illustrated by the following tables.

**Table 11.** Without  $A_t$  no  $B_t$

		$B_t$		
		TRUE	FALSE	
$A_t$	TRUE	$a_t$	$b_t$	$A_t$
	FALSE	$c_t = 0$	$d_t$	$\underline{A}_t$
		$B_t$	$\underline{B}_t$	+1

**Table 12.** If  $B_t$  then  $A_t$

		$A_t$		
		TRUE	FALSE	
$B_t$	TRUE	$a_t$	$c_t = 0$	$B_t$
	FALSE	$b_t$	$d_t$	$\underline{B}_t$
		$A_t$	$\underline{A}_t$	+1

There are circumstances under which

$$p(A_t \leftarrow B_t) \equiv \underbrace{(A_t \vee B_t)}_{\text{(Necessary condition)}} \equiv \underbrace{(\underline{A}_t \vee B_t)}_{\text{(Sufficient condition)}} \equiv p(A_t \rightarrow B_t) \quad (296)$$

However, equation 295 does not imply the relationship of equation 296 under any circumstances.

### Example I.

A wax candle is characterised by various properties, but is also subject to certain conditions. **Without** sufficient amounts of gaseous oxygen **no** burning wax candle, gaseous oxygen is a necessary condition of a burning candle. However, the converse relationship **if** burning wax candle, **then** sufficient amounts of gaseous oxygen are given is at the same (period of) time  $t$  / Bernoulli trial  $t$  true. The following tables are illustrating these relationships.

**Table 13.** Without gaseous oxygen no burning candle

		Burning candle		
		TRUE	FALSE	
Gaseous oxygen	TRUE	$a_t$	$b_t$	$A_t$
	FALSE	$c_t = 0$	$d_t$	$\underline{A}_t$
		$B_t$	$\underline{B}_t$	+1

**Table 14.** If burning candle then gaseous oxygen

		Gaseous oxygen		
		TRUE	FALSE	
Burning candle	TRUE	$a_t$	$c_t = 0$	$B_t$
	FALSE	$b_t$	$d_t$	$\underline{B}_t$
		$A_t$	$\underline{A}_t$	+1

### Example II.

Once again, a human being cannot live without water. A human being cannot live without gaseous oxygen, et cetera. Water itself is a necessary condition for human life. However, gaseous oxygen is a necessary condition for human life too. Thus far, even if water is given and even if water is a necessary condition for human life, without gaseous oxygen there will be no human life. In general, if a conditioned or an outcome  $B_t$  depends on the necessary condition  $A_t$  and equally on numerous other

necessary conditions, an event  $B_t$  will not occur if  $A_t$  itself is not given independently of the occurrence of other necessary conditions.

### Example III.

Another different aspect of a necessary condition relationship is appropriate to be focused upon here. As a direct consequence of a necessary condition **without** sufficient amounts of gaseous oxygen **no** burning wax candle is a special case of an exclusion relationship. The absence of sufficient amounts of gaseous oxygen  $A_t$  excludes (see Barukčić, 2021a) a burning wax candle  $B_t$ . Thus far, if we want to stop the burning of a wax candle, we would have to significantly reduce the amounts of gaseous oxygen  $A_t$ . Under these conditions, a wax candle will stop burning. The following tables (table 15 and table 16) may illustrate this aspect of a necessary condition in more detail.

**Table 15.** Without gaseous oxygen no burning candle

		Burning candle		
		TRUE	FALSE	
Gaseous oxygen	TRUE	$a_t$	$b_t$	$A_t$
	FALSE	$c_t = 0$	$d_t$	$\underline{A}_t$
		$B_t$	$\underline{B}_t$	+1

**Table 16.** Absent gaseous oxygen excludes burning wax candle

		Burning candle		
		TRUE	FALSE	
Gaseous oxygen	FALSE	$c_t = 0$	$d_t$	$B_t$
	TRUE	$a_t$	$b_t$	$\underline{B}_t$
		$A_t$	$\underline{A}_t$	+1

The necessary condition relationship follows approximately (see Barukčić, 2022b) as

$$p(A_t \leftarrow B_t) \geq 1 - \frac{p(c_t)}{p(B_t)} \quad (297)$$

and as

$$p(A_t \leftarrow B_t) \geq 1 - \frac{p(c_t)}{p(\underline{A}_t)} \quad (298)$$

#### 2.3.10. The Chi-square goodness of fit test of a necessary condition relationship

##### Definition 2.44 (The $\tilde{\chi}^2$ goodness of fit test of a necessary condition relationship).

Under some well known circumstances, hypothesis about the *conditio sine qua non* relationship  $p(A_t \leftarrow B_t)$  can be tested by the chi-square distribution (also chi-squared or  $\chi^2$ -distribution), first described by the German statistician Friedrich Robert Helmert (Helmert, 1876) and later rediscovered by Karl Pearson (Pearson, 1900a) in the context of a goodness of fit test. The  $\tilde{\chi}^2$  goodness of fit test of a *conditio sine qua non* relationship with degree of freedom (d. f.) of d. f. = 1 is calculated as

$$\begin{aligned}
 \tilde{\chi}^2_{\text{Calculated}}(A_t \leftarrow B_t | B) &\equiv \frac{(a - (a + c))^2}{B} + \frac{((b + d) - \underline{B})^2}{\underline{B}} \\
 &\equiv \frac{c^2}{B} + 0 \\
 &\equiv \frac{c^2}{B}
 \end{aligned} \tag{299}$$

or equally as

$$\begin{aligned}
 \tilde{\chi}^2_{\text{Calculated}}(A_t \leftarrow B_t | A) &\equiv \frac{(d - (c + d))^2}{A} + \frac{((a + b) - A)^2}{A} \\
 &\equiv \frac{c^2}{A} + 0 \\
 &\equiv \frac{c^2}{A}
 \end{aligned} \tag{300}$$

and can be compared with a theoretical chi-square value at a certain level of significance  $\alpha$ . It has not yet been finally clarified whether the use of Yate's (Yates, 1934) continuity correction is necessary at all.

### 2.3.11. The left-tailed p Value of the conditio sine qua non relationship

#### **Definition 2.45 (The left-tailed p Value of the conditio sine qua non relationship).**

The left-tailed (lt) p Value (Barukčić, 2019e) of the conditio sine qua non relationship can be calculated as follows.

$$\begin{aligned}
 pValue_{lt}(A_t \leftarrow B_t) &\equiv 1 - e^{-(1-p(A_t \leftarrow B_t))} \\
 &\equiv 1 - e^{-(c/N)}
 \end{aligned} \tag{301}$$

## 2.3.12. Sufficient condition

**Definition 2.46** (Sufficient condition [*Conditio per quam*]).

Mathematically, the sufficient (Barukčić, 2021c, p. 68-70) condition (see Barukčić, 2022a) (IMP) relationship, denoted by  $p(A_t \rightarrow B_t)$  in terms of statistics and probability theory, is defined (Barukčić, 1989, p. 68-70) as

$$\begin{aligned}
 p(A_t \rightarrow B_t) &\equiv p(\underline{A}_t \vee B_t) \equiv \frac{\sum_{t=1}^N (\underline{A}_t \vee B_t)}{N} \equiv \frac{(\underline{A}_t \vee B_t) \times p(\underline{A}_t \vee B_t)}{(\underline{A}_t \vee B_t)} \\
 &\equiv p(a_t) + p(c_t) + p(d_t) \\
 &\equiv \frac{N \times (p(a_t) + p(c_t) + p(d_t))}{N} \\
 &\equiv \frac{a + c + d}{N} \equiv \frac{E(\underline{A}_t \vee B_t)}{N} \\
 &\equiv \frac{B + d}{N} \equiv \frac{E(A_t \rightarrow B_t)}{N} \\
 &\equiv \frac{a + A}{N} \\
 &\equiv +1
 \end{aligned} \tag{302}$$

In general, it is  $p(A_t \succ B_t) \equiv 1 - p(A_t \rightarrow B_t)$  (see Table 17).

**2.3.12.1. Mackie's INUS Condition** John Leslie Mackie (1917-1981) critically examined the theories of causation of various (see Ducasse, 1926) philosophers such as Hume (Book I, Part III, of the Treatise) (see Mackie, 1974, pp. 3-28), Kant (as well as Kantian approaches offered by Strawson and Bennett), Mill and other. Mackie rightly claims that Hume's regularity theory of causation offer only an incomplete picture of the nature of causation. Mackie writes: "It seems appropriate to begin by examining and criticizing it, so that we can take over from it whatever seems to be defensible but develop an improved account by correcting its errors and deficiencies." (see Mackie, 1974, p. 3). Nonetheless, in his trial to develop an improved account of Hume's theory of causation, Mackie's own account of the nature of causation follows Hume's principles of causation very closely (see Mackie, 1974, pp. 3-28). Mackie himself proposed already in 1965 that "the so-called cause is ... an *insufficient* but *necessary* part of a condition which is itself *unnecessary* but *sufficient* for the result ... let us call such a condition ... an INUS condition." (see Mackie, 1965, p. 245). However Mackie's account needs modification, and can be modified and when it is modified we can explain much more satisfactorily what Mackie ordinarily take to be a cause. Mackie is of the opinion that "... cause is ... part of a condition ... " (see Mackie, 1965, p. 245) and that "... a condition ... is ... *unnecessary* but *sufficient* for the result [i. e. effect, author]. " (see Mackie, 1965, p. 245). To put it very simply one could say that Mackie reduces a cause to a sufficient condition, "... cause is ... a condition which is itself ... *sufficient* ... " (see Mackie, 1965, p. 245). Indeed, there are circumstances, where several

different events <sup>104</sup> might be necessary or sufficient et cetera at the same time in order to determine **a compound/complex sufficient condition relationship**. Thus far, it seems appropriate to take over from Mackie's INUS condition whatever seems to be acceptable but to develop an improved account by correcting its deficiencies and errors in order to do justice to the complexity of affairs. Equation 303 illustrates one real-world example of a compound/complex sufficient condition relationship in more detail.

$$\begin{aligned}
 p(((X_1 \wedge X_2 \wedge X_3 \wedge \dots) \wedge A_t) \rightarrow B_t) &\equiv p(\underbrace{((X_1 \wedge X_2 \wedge X_3 \wedge \dots) \wedge A_t)} \vee B_t) \\
 &\equiv \frac{\sum_{t=1}^N \left( \underbrace{((X_1 \wedge X_2 \wedge X_3 \wedge \dots) \wedge A_t)} \vee B_t \right)}{N} \\
 &\equiv +1
 \end{aligned} \tag{303}$$

Again, taking into account Kolmogorov's definition of an n-dimensional probability density (see also Kolmogorov, Andreĭ Nikolaevich, 1950, p. 26) of random variables  $A_t$ ,  $B_t$  et cetera at the (period of) time  $t$ , we obtain

$$\begin{aligned}
 p(A_t \rightarrow B_t) &\equiv +1 \\
 &\equiv +1 - p(\underline{B}_t) \\
 &\equiv +1 - p(A_t \cap \underline{B}_t) \\
 &\equiv \left( \int_{-\infty}^{A_t} \int_{-\infty}^{B_t} f(A_t, B_t) dA_t dB_t \right) + \left( 1 - \int_{-\infty}^{A_t} f(A_t) dA_t \right)
 \end{aligned} \tag{304}$$

while  $p(A_t \rightarrow B_t)$  would denote the cumulative distribution function of random variables of a sufficient condition. Another adequate formulation of a sufficient condition is possible too.

**Table 17.** Sufficient condition.

		Conditioned $B_t$		
		TRUE	FALSE	
Condition	TRUE	$p(a_t)$	<b>+0</b>	$p(A_t)$
	$A_t$	FALSE	$p(c_t)$	$p(\underline{A}_t)$
		$p(B_t)$	$p(\underline{B}_t)$	+1

**Remark 2.3.** A sufficient condition  $A_t$  is characterized by the property that another event  $B_t$  will occur if  $A_t$  is given, if  $A_t$  itself occurred (Barukčić, 1989, 1997, 2005, 2016b, 2017b,c, 2020a,b,c,d, Barukčić and Ufuoma, 2020). **Example.** The ground, the streets, the trees, human beings and many other objects too will become wet during heavy rain. Especially, **if** it is raining (event  $A_t$ ), **then** human beings will become wet (event  $B_t$ ). However, even if this is a common human wisdom, a human being equipped with an appropriate umbrella (denoted by  $R_t$ ) need not become wet even during heavy rain. An appropriate umbrella ( $R_t$ ) is similar to an event with the potential to counteract the occurrence of another event

<sup>104</sup>Barukčić, Ilija. (2022). *Conditio per quam*. *Causation*, 17(3), 5–86. <https://doi.org/10.5281/zenodo.6369831>



$(B_t)$  and can be understood something as an **anti-dot** of another event. In other words, an appropriate umbrella is an antidote of the effect of rain on human body, an appropriate umbrella has the potential to protect humans from the effect of rain on their body. It is a good rule of thumb that the following relationship

$$p(A_t \rightarrow B_t) + p(R_t \wedge B_t) \equiv +1 \quad (305)$$

indicates that  $R_t$  is an antidote of  $A_t$ . However, taking a shower, swimming in a lake et cetera may make human hair wet too. More than anything else, however, these events does not affect the final outcome, the effect of raining on human body.

The approximate (see Barukčić, 2022a) value of the material implication is given as

$$p(A_t \rightarrow B_t) \geq 1 - \frac{p(b_t)}{p(A_t)} \quad (306)$$

and alternatively as

$$p(A_t \rightarrow B_t) \geq 1 - \frac{p(b_t)}{p(\underline{B}_t)} \quad (307)$$

### 2.3.13. The Chi square goodness of fit test of a sufficient condition relationship

**Definition 2.47 (The  $\tilde{\chi}^2$  goodness of fit test of a sufficient condition relationship).**

Under some well known circumstances, testing hypothesis about the conditio per quam relationship  $p(A_t \rightarrow B_t)$  is possible by the chi-square distribution (also chi-squared or  $\tilde{\chi}^2$ -distribution) too. The  $\tilde{\chi}^2$  goodness of fit test of a conditio per quam relationship with degree of freedom (d. f.) of d. f. = 1 is calculated as

$$\begin{aligned} \tilde{\chi}^2_{\text{Calculated}}(A_t \rightarrow B_t | A) &\equiv \frac{(a - (a+b))^2}{A} + \frac{((c+d) - \underline{A})^2}{\underline{A}} \\ &\equiv \frac{b^2}{A} + 0 \\ &\equiv \frac{b^2}{A} \end{aligned} \quad (308)$$

or equally as

$$\begin{aligned}
 \tilde{\chi}^2_{\text{Calculated}}(A_t \rightarrow B_t | \underline{B}) &\equiv \frac{(d - (b + d))^2}{\underline{B}} + \\
 &\quad \frac{((a + c) - B)^2}{B} \\
 &\equiv \frac{b^2}{\underline{B}} + 0 \\
 &\equiv \frac{b^2}{\underline{B}}
 \end{aligned} \tag{309}$$

and can be compared with a theoretical chi-square value at a certain level of significance  $\alpha$ . The  $\tilde{\chi}^2$ -distribution equals zero when the observed values are equal to the expected/theoretical values of the conditio per quam relationship/distribution  $p(A_t \rightarrow B_t)$ , in which case the null hypothesis is accepted. Yate's (Yates, 1934) continuity correction has not been used in this context.

2.3.14. The left-tailed p Value of the conditio per quam relationship

**Definition 2.48 (The left-tailed p Value of the conditio per quam relationship).**

The left-tailed (lt) p Value (Barukčić, 2019e) of the conditio per quam relationship can be calculated as follows.

$$\begin{aligned}
 pValue_{lt}(A_t \rightarrow B_t) &\equiv 1 - e^{-(1-p(A_t \rightarrow B_t))} \\
 &\equiv 1 - e^{-(b/N)}
 \end{aligned} \tag{310}$$

Again, a low p-value indicates a statistical significance.

2.3.15. Necessary and sufficient conditions

**Definition 2.49 (Necessary and sufficient conditions [EQV]).**

The necessary and sufficient condition (EQV) relationship, denoted by  $p(A_t \leftrightarrow B_t)$  in terms of

statistics and probability theory, is defined (Barukčić, 1989, p. 68-70) as

$$\begin{aligned}
 p(A_t \leftrightarrow B_t) &\equiv \frac{\sum_{t=1}^N ((A_t \vee \underline{B}_t) \wedge (\underline{A}_t \vee B_t))}{N} \\
 &\equiv p(a_t) + p(d_t) \\
 &\equiv \frac{N \times (p(a_t) + p(d_t))}{N} \\
 &\equiv \frac{a + d}{N} \\
 &\equiv +1
 \end{aligned} \tag{311}$$

2.3.16. The Chi square goodness of fit test of a necessary and sufficient condition relationship

**Definition 2.50 (The  $\tilde{\chi}^2$  goodness of fit test of a necessary and sufficient condition relationship).**

Even the necessary and sufficient condition relationship  $p(A_t \leftrightarrow B_t)$  can be tested by the chi-square distribution (also chi-squared or  $\tilde{\chi}^2$ -distribution) too. The  $\tilde{\chi}^2$  goodness of fit test of a necessary and sufficient condition relationship with degree of freedom (d. f.) of d. f. = 1 is calculated as

$$\begin{aligned}
 \tilde{\chi}^2_{\text{Calculated}}(A_t \leftrightarrow B_t | A) &\equiv \frac{(a - (a+b))^2}{A} + \\
 &\quad \frac{d - ((c+d))^2}{\underline{A}} \\
 &\equiv \frac{b^2}{A} + \frac{c^2}{\underline{A}}
 \end{aligned} \tag{312}$$

or equally as

$$\begin{aligned}
 \tilde{\chi}^2_{\text{Calculated}}(A_t \leftrightarrow B_t | B) &\equiv \frac{(a - (a+c))^2}{B} + \\
 &\quad \frac{d - ((b+d))^2}{\underline{B}} \\
 &\equiv \frac{c^2}{B} + \frac{b^2}{\underline{B}}
 \end{aligned} \tag{313}$$

The calculated  $\tilde{\chi}^2$  goodness of fit test of a necessary and sufficient condition relationship can be compared with a theoretical chi-square value at a certain level of significance  $\alpha$ . Under conditions where the observed values are equal to the expected/theoretical values of a necessary and sufficient condition relationship/distribution  $p(A_t \leftrightarrow B_t)$ , the  $\tilde{\chi}^2$ -distribution equals zero. It is to be cleared whether Yate's (Yates, 1934) continuity correction should be used at all.

## 2.3.17. The left-tailed p Value of a necessary and sufficient condition relationship

**Definition 2.51 (The left-tailed p Value of a necessary and sufficient condition relationship).**

The left-tailed (lt) p Value (Barukčić, 2019e) of a necessary and sufficient condition relationship can be calculated as follows.

$$\begin{aligned} pValue_{lt}(A_t \leftrightarrow B_t) &\equiv 1 - e^{-(1-p(A_t \leftrightarrow B_t))} \\ &\equiv 1 - e^{-((b+c)/N)} \end{aligned} \quad (314)$$

In this context, a low p-value indicates again a statistical significance. Table 18 may provide an overview of the theoretical distribution of a necessary and sufficient condition.

**Table 18.** Necessary and sufficient condition.

		Conditioned $B_t$		
		YES	NO	
Condition $A_t$	YES	1	0	1
	NO	0	1	1
		1	1	2

## 2.3.18. Either or conditions

**Definition 2.52 (Either  $A_t$  or  $B_t$  conditions [NEQV]).**

Mathematically, an either  $A_t$  or  $B_t$  condition relationship (NEQV), denoted by  $p(A_t \succ\prec B_t)$  in terms of statistics and probability theory, is defined (Barukčić, 1989, p. 68-70) as

$$\begin{aligned} p(A_t \succ\prec B_t) &\equiv \frac{\sum_{t=1}^N ((A_t \wedge \underline{B}_t) \vee (\underline{A}_t \wedge B_t))}{N} \\ &\equiv p(b_t) + p(c_t) \\ &\equiv \frac{N \times (p(b_t) + p(c_t))}{N} \\ &\equiv \frac{b+c}{N} \\ &\equiv +1 \end{aligned} \quad (315)$$

It is  $p(A_t \succ\prec B_t) \equiv 1 - p(A_t \leftrightarrow B_t)$  (see Table 19).

**Table 19.** Either  $A_t$  or  $B_t$  relationship.

		Conditioned $B_t$		
		YES	NO	
Condition $A_t$	YES	0	1	1
	NO	1	0	1
		1	1	2

### 2.3.19. The Chi-square goodness of fit test of an either or condition relationship

#### **Definition 2.53 (The $\tilde{\chi}^2$ goodness of fit test of an either or condition relationship).**

An either or condition relationship  $p(A_t \succ\prec B_t)$  can be tested by the chi-square distribution (also chi-squared or  $\tilde{\chi}^2$ -distribution) too. The  $\tilde{\chi}^2$  goodness of fit test of an either or condition relationship with degree of freedom (d. f.) of d. f. = 1 is calculated as

$$\begin{aligned} \tilde{\chi}^2_{\text{Calculated}}((A_t \succ\prec B_t) | A) &\equiv \frac{(b - (a + b))^2}{A} + \frac{c - ((c + d))^2}{\frac{A}{B}} \\ &\equiv \frac{a^2}{A} + \frac{d^2}{\frac{A}{B}} \end{aligned} \quad (316)$$

or equally as

$$\begin{aligned} \tilde{\chi}^2_{\text{Calculated}}((A_t \succ\prec B_t) | B) &\equiv \frac{(c - (a + c))^2}{B} + \frac{b - ((b + d))^2}{\frac{B}{A}} \\ &\equiv \frac{a^2}{B} + \frac{d^2}{\frac{B}{A}} \end{aligned} \quad (317)$$

Yate's (Yates, 1934) continuity correction has not been used in this context.

### 2.3.20. The left-tailed p Value of an either or condition relationship

#### **Definition 2.54 (The left-tailed p Value of an either or condition relationship).**

---

The left-tailed (lt) p Value (Barukčić, 2019e) of an either or condition relationship can be calculated as follows.

$$\begin{aligned} pValue_{lt}(A_t \succ\prec B_t) &\equiv 1 - e^{-(1-p(A_t \succ\prec B_t))} \\ &\equiv 1 - e^{-((a+d)/N)} \end{aligned} \quad (318)$$

In this context, a low p-value indicates again a statistical significance.

## 2.4. Causation

### 2.4.1. Causation in general

The history of the denialism of causality in Philosophy, Mathematics, Statistics, Physics et cetera is very long. We only recall David Hume's (1711-1776) account of causation and his inappropriate reduction of the cause-effect relationship to a simple habitual connection in human thinking or Immanuel Kant's (1724-1804) initiated trial to consider causality as nothing more but a '*a priori*' given category (Langsam, 1994) in human reasoning and other similar attempts too.

It is worth noting in this context that especially Karl Pearson (1857 - 1936) himself has been engaged in a long lasting and never-ending crusade against causation too. **"Pearson categorically denies the need for an independent concept of causal relation beyond correlation ... he exterminated causation from statistics before it had a chance to take root"** (see Pearl, 2000, p. 340).

At the beginning of the 20<sup>th</sup> century notable proponents of **conditionalism** like the German anatomist and pathologist David Paul von Hansemann (Hansemann, David Paul von, 1912) (1858 - 1920) and the biologist and physiologist Max Richard Constantin Verworn (Verworn, 1912) (1863 - 1921) started a new attack (Kröber, 1961) on the principle of causality. In his essay "Kausale und konditionale Weltanschauung" Verworn (Verworn, 1912) presented "an exposition of 'conditionism' as contrasted with 'causalism,'" (Unknown, 1913) while ignoring cause and effect relationships completely. **"Das Ding ist also identisch mit der Gesamtheit seiner Bedingungen."** (Verworn, 1912) However, Verworn's goal to exterminate causality completely out of science was hindered by the further development of research.

The history of futile attempts to refute **the principle of causality** culminated in a publication by the German born physicist Werner Karl Heisenberg (1901 - 1976). Heisenberg put forward an illogical, inconsistent and confusing uncertainty principle which opened the door to wishful thinking and logical fallacies in physics and in science as such. Heisenberg's unjustified reasoning ended in an act of a manifestly unfounded conclusion: **"Weil alle Experimente den Gesetzen der Quantenmechanik und damit der Gleichung (1) unterworfen sind, so wird durch die Quantenmechanik die Ungültigkeit des Kausalgesetzes definitiv festgestellt."** (Heisenberg, Werner Karl, 1927) while 'Gleichung (1)' denotes Heisenberg's uncertainty principle. Einstein's himself, a major contributor to quantum theory and in the same respect a major critic of quantum theory, disliked Heisenberg's uncertainty principle fundamentally while Einstein's opponents used Heisenberg's Uncertainty Principle against Einstein. After the End of the German Nazi initiated Second World War with unimaginable brutality and high human losses and a death toll due to an industrially organised mass killing of people by the German Nazis which did not exist in this way before, Werner Heisenberg visited Einstein in Princeton (New Jersey, USA) in October 1954 (Neffe, 2006). Einstein agreed to meet Heisenberg only for a very short period of time but their encounter lasted longer. However, there were not only a number of differences between Einstein and Heisenberg, these two physicists did not really love each other. "Einstein remarked that the inventor of the uncertainty principle was a 'big Nazi'..." (Neffe, 2006) Albert Einstein (1879 - 1955) took again the opportunity to refuse to endorse **Heisenberg's uncertainty principle**

as a fundamental law of nature and rightly too. Meanwhile, Heisenberg's uncertainty principle is refuted (see Barukčić, 2011a, 2014, 2016a) for several times but still not exterminated completely out of physics and out of science as such.

In contrast to such extreme anti-causal positions as advocated by Heisenberg and the **Copenhagen interpretation of quantum mechanics**, the search for a (mathematical) solution of *the issue of causal inferences* is as old as human mankind itself (“*i. e. Aristotle's Doctrine of the Four Causes*”) (Hennig, 2009) even if there is still little to go on.

It is appropriate to specify especially the position of D'Holbach (Holbach, Paul Henri Thiry Baron de, 1770). D'Holbach (1723–1789) himself linked cause and effect or causality as such to changes. “**Une cause, est un être qui e met un autre en mouvement, ou qui produit quelque changement en lui. L'effet est le changement qu'un corps produit dans un autre ...**” (Holbach, Paul Henri Thiry Baron de, 1770). D'Holbach infers in the following: “**De l'action et de la réaction continuelle de tous les êtres que la nature renferme, il résulte une suite de causes et d'effets ...**” (Holbach, Paul Henri Thiry Baron de, 1770).

With more or less meaningless or none progress on the matter in hand even in the best possible conditions, it is not surprising that authors are suggesting more and more different approaches and models for causal inference. Indeed, the hope is justified that logically consistent *statistical methods of causal inference* can help scientist to achieve so much with so little.

One of the methods of causal inference in Bio-sciences are based on the known *Henle* (Henle, 1840) (1809–1885) - *Koch* (Koch, 1878) (1843–1910) postulates (Carter, 1985) which are applied especially for the identification of a causative agent of an (infectious) disease. However, the pathogenesis of most chronic diseases is more or less very complex and potentially involves the interaction of several factors. In practice, from the ‘pure culture’ requirement of the Henle-Koch postulates insurmountable difficulties may emerge. In light of subsequent developments (PCR methodology, immune antibodies et cetera) it is appropriate to review the full validity of the Henle-Koch postulates in our days.

In 1965, Sir Austin Bradford Hill (Hill, 1965) published nine criteria (the ‘*Bradford Hill Criteria*’) in order to determine whether observed epidemiological associations are causal. Somewhat worrying, is at least the fact that, Hill's “... fourth characteristic is *the temporal relationship of the association*” and so-to-speak just a reformulation of the ‘*post hoc ergo propter hoc*’ (Barukčić, 1989, Woods and Walton, 1977) logical fallacy through the back-door and much more than this. It is questionable whether association as such can be treated as being identical with causation. Unfortunately, due to several reasons, it seems therefore rather problematic to rely on Bradford Hill Criteria carelessly.

Meanwhile, several other and competing mathematical or statistical approaches for causal inference have been discussed by various modern authors (Barukčić, 1989, 1997, 2005, 2016b, 2017a,c, Bohr, 1937, Chisholm, 1946, Dempster, 1990, Espejo, 2007, Goodman, 1947, Granger, 1969, Hessen, Johannes, 1928, Hesslow, 1976, 1981, Korch, Helmut, 1965, Lewis, David Kellogg, 1973, 1974, Pearl, 2000, Schlick, Friedrich Albert Moritz, 1931, Spohn, 1983, Suppes, 1970, Todd, 1968, Zesar, 2013) or even established (Barukčić, 1989, 1997, 2005, 2016b, 2017a,c). Nevertheless, the question is still



not answered, is it at all possible to establish a cause effect relationship between two factors while applying only certain statistical (Sober, 2001) methods?

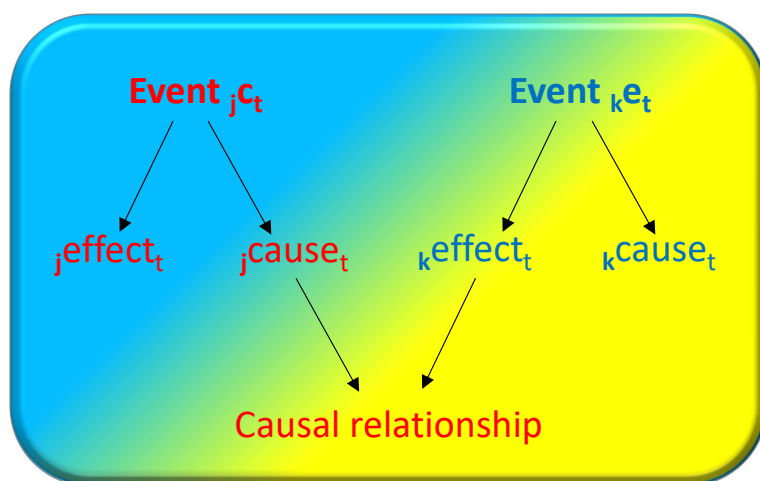
## 2.4.2. Cause and effect

Besides all, there are several further aspects of causation for which our attention so far has not been adequately fixed in this context. In the causal relationship, cause and effect are united, a cause is an effect and an effect is a cause.

“Thus, in the causal relation, cause and effect are inseparable; a cause which had no effect would not be a cause, just as an effect which had no cause would no longer be an effect. ”

(see Hegel, Georg Wilhelm Friedrich, 1991, p. 151)

The unity of cause and effect is a unity of two which are not the same. Cause and effect as inseparable in the causal relation are at the same time mutually related as sheer others; each of both as united in its own self to the other of itself is able to pass over into its own other and vice versa. Yet, to approach from a different point of view, a cause and an effect are separated in the same relation too, a cause is not an effect and an effect is not a cause, both are different in the same relation.



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“Therefore, though the cause has an effect and is at the same time itself effect, and the effect not only has a cause but is also itself cause, yet the effect which the cause has, and the effect which it is, are different, as are also the cause which the effect has, and the cause which it is.” (see Hegel, Georg Wilhelm Friedrich, 1991, p. 565/566)

**2.4.2.1. What is a cause, what is an effect?** An important fact to which we must pay attention here is that in a causal relation, under certain circumstances, an individual cause and an individual effect are related to each other in their own particular way. An effect which vanishes in its own cause in the same respect equally becomes again in it and vice versa. A cause which is merely extinguished in its own effect becomes again in the same. In fact, each of these determinations presupposes in its own other its own self and constitutes the intimate tie between an individual cause and its own individual

effect. Thus far, under conditions of a **positive** causal relationship  $k$ , an event  $U_t$  which is for sure a cause of another event  $W_t$  is at the same time  $t$  a necessary and sufficient condition of an event  $W_t$ . Table 20 may illustrate this relationship. A matter of great theoretical importance is the fundamental

**Table 20.** What is the cause, what is the effect?

		Effect $W_t$		
		TRUE	FALSE	
Cause	TRUE	<b>+1</b>	<b>+0</b>	$p(U_t)$
$U_t$	FALSE	<b>+0</b>	<b>+1</b>	$p(\underline{U}_t)$
		$p(W_t)$	$p(\underline{W}_t)$	+1

relationship between a cause and a condition. Are both, cause and condition, at the end identical? As of now, following Mill (see [Mill, 1843a](#), p. 403), Verworn (see [Verworn, 1912](#)), Mackie and others, we can give a clear ‘Yes’ in reply to this question: “... cause is ... a condition which is itself ... *sufficient* ... ” (see [Mackie, 1965](#), p. 245 ). However, this issue is not as simple as it sounds, according to Mackie. Thus far, it is essential to eliminate some errors. Indeed, there are circumstances where a cause and a condition are identical, a cause and a condition are equivalent. However, as outlined in this publication, both, a cause and a condition, are different too and a cause and a condition are not identical either.

“Jede Ursache ist nothwendig auch eine Bedingung eines Ereignisses;  
aber nicht jede Bedingung ist Ursache zu nennen. ”

(see [Bar, Carl Ludwig von, 1871](#), p. 4)

The crux of the matter is that not every condition is a cause too, in German: “... nicht jede Bedingung ist Ursache ... ”(see [Bar, Carl Ludwig von, 1871](#), p. 4). However, and in contrast to a condition, every cause as such is indeed a condition too, in German: “Jede Ursache ist ... auch eine Bedingung ... ”(see [Bar, Carl Ludwig von, 1871](#), p. 4). In general, a cause  $U_t$  is a necessary condition of an effect  $W_t$ . In other words, **without** a cause  $U_t$  **no** effect  $W_t$ . One consequence of the necessary condition relationship between cause and effect is that “... an effect which had no cause would no longer be an effect. ” (see [Hegel, Georg Wilhelm Friedrich, 1991](#), p. 151). However, a cause  $U_t$  being a necessary condition of an effect  $W_t$  is equivalent to an effect  $W_t$  being a sufficient condition of the same cause  $U_t$  and vice versa too. In our everyday words,

**without**

$U_t$

**no**

$W_t$

is equivalent with

**if**

$W_t$

**then**

$U_t$

and vice versa. As can be seen, there is a kind of strange mirroring between  $U_t$  and  $W_t$  at the same Bernoulli trial  $t$ . Lastly, both are converses of each other too. In other words,  $U_t$ 's being a necessary condition of  $W_t$ 's is equivalent to  $W_t$ 's being a sufficient condition of  $U_t$ 's (and vice versa). In general, it is

$$(U_t \vee \underline{W}_t) \equiv (\underline{W}_t \vee U_t) \equiv ((U_t \vee \underline{W}_t) \wedge (\underline{W}_t \vee U_t)) \equiv +1 \quad (319)$$

		Effect $W_t$		
		TRUE	FALSE	
Cause	TRUE	$a_t$	$b_t$	$U_t$
$U_t$	FALSE	$c_t = 0$	$d_t$	$\underline{U}_t$
		$W_t$	$\underline{W}_t$	+1

**Table 21.** Without  $U_t$  no  $W_t$

		Cause $U_t$		
		TRUE	FALSE	
Effect	TRUE	$a_t$	$c_t = 0$	$W_t$
$W_t$	FALSE	$b_t$	$d_t$	$\underline{W}_t$
		$U_t$	$\underline{U}_t$	+1

**Table 22.** If  $W_t$  then  $U_t$

The other side of the causal relation at the same (period of) time / Bernoulli trial  $t$  is the fact that a cause  $U_t$  is equally a sufficient condition of an effect  $W_t$  too or shortly **if** cause  $U_t$  **then** effect  $W_t$ . One straightforward consequence of this fundamental relationship between a cause and an effect is that "... a cause which had no effect would not be a cause ... " (see [Hegel, Georg Wilhelm Friedrich, 1991](#), p. 151). But even this is not without difficulties, because a cause  $U_t$  being a sufficient condition of an effect  $W_t$  is equivalent to effect  $W_t$  being a necessary condition of the same cause  $U_t$ . In different words,

**if**

$U_t$

**then**

$W_t$

is equivalent with

**without**

$W_t$

**no**

$U_t$ .

		Effect $W_t$		$U_t$
		TRUE	FALSE	
Cause	TRUE	$a_t$	$b_t = 0$	$U_t$
$U_t$	FALSE	$c_t$	$d_t$	$\underline{U}_t$
		$W_t$	$\underline{W}_t$	+1

**Table 23.** If  $U_t$  then  $W_t$

		Cause $U_t$		$W_t$
		TRUE	FALSE	
Effect	TRUE	$a_t$	$c_t$	$W_t$
$W_t$	FALSE	$b_t = 0$	$d_t$	$\underline{W}_t$
		$U_t$	$\underline{U}_t$	+1

**Table 24.** Without  $W_t$  no  $U_t$

To bring it to the point, necessary and sufficient conditions are at the end converses (see [Gomes, Gilberto, 2009](#)) of each other and far more than this. In fact, there is a kind of reciprocity or mirroring between cause and effect. Necessary and sufficient conditions are relationships used to describe the relationship between two events at the same Bernoulli trial  $t$ . In more detail, if  $U_t$  then  $W_t$  is equivalent with  $W_t$  is necessary for  $U_t$ , because the truth of  $U_t$  guarantees the truth of  $W_t$ . In general, it is

$$(\underline{U}_t \vee W_t) \equiv (W_t \vee \underline{U}_t) \equiv ((\underline{U}_t \vee W_t) \wedge (W_t \vee \underline{U}_t)) \equiv +1 \quad (320)$$

In other words, it is impossible to have  $U_t$  without  $W_t$  ([Bloch, 2011](#)). Similarly,  $U_t$  is sufficient for  $W_t$ , because  $U_t$  being true always implies that  $W_t$  is true, but  $U_t$  not being true does not always imply that  $W_t$  is not true. And we should use this relationships to make our point. In general, **without** gaseous oxygen ( $U_t$ ), there is **no** burning wax candle ( $W_t$ ); hence the relationship **if** burning wax candle ( $W_t$ ) **then** gaseous oxygen ( $U_t$ ) is equally true and given. This everyday knowledge is known and secured since centuries and might be illustrated as follows.

		Wax candle $B_t$		$A_t$
		burning	not burning	
Gaseous oxygen <sub>t</sub>	present	$a_t$	$b_t$	$A_t$
	not present	$c_t = 0$	$d_t$	$\underline{A}_t$
		$B_t$	$\underline{B}_t$	+1

**Table 25.** Without  $A_t$  no  $B_t$

		Gaseous oxygen $A_t$		$B_t$
		present	not present	
Wax candle	burning	$a_t$	$c_t = 0$	$B_t$
	not burning	$b_t$	$d_t$	$\underline{B}_t$
		$A_t$	$\underline{A}_t$	+1

**Table 26.** If  $B_t$  then  $A_t$

Nonetheless, and independently of this secured everyday knowledge, **a burning wax candle is a sufficient condition of gaseous oxygen but not the cause of gaseous oxygen.**

Given all the circumstances, it is at least this simple **counter-example** which provides us with a convincing evidence that **a sufficient condition alone is not enough to describe a cause completely.** In general, a cause as such cannot be reduced to a simple sufficient condition.

In contrast to this obvious fact, other authors prefer another approach to the definition of a cause. “So that, more explicitly, if a given particular event is regarded as having been sufficient to the occurrence of another, it is said to have been its cause; if regarded as having been necessary to the occurrence of another, it is said to have been a condition of it; ...” (see [Ducasse, 1926](#), p. 58). Therefore, in order

to be a cause of oxygen, additional evidence is necessary that a burning wax candle is a necessary condition of gaseous oxygen too. However, even if the relationship **without** gaseous oxygen **no** burning wax candle is given, this relationship is not given vice versa. The relationship **without** burning wax candle **no** gaseous oxygen is not given. Like other fundamental concepts, the concepts of cause and effect can be associated with difficulties too. Under certain conditions, the causal relationship between  $U_t$  and  $W_t$ , when correctly defined and recognised, is closely allied with the requirement that a certain study or that at least other, different studies provided evidence of a necessary condition between  $U_t$  and  $W_t$  and of a sufficient condition between  $U_t$  and  $W_t$  and if possible of a **necessary and sufficient condition** between  $U_t$  and  $W_t$  too.

Mathematically, a necessary and sufficient condition between  $U_t$  and  $W_t$  is defined as

$$(U_t \vee \underline{W}_t) \wedge (\underline{U}_t \vee W_t) \equiv +1 \quad (321)$$

However, I think it necessary to make a clear distinction between a necessary and sufficient condition and the converse relationship (Eq. 319) above.

$$((U_t \vee \underline{W}_t) \wedge (\underline{W}_t \vee U_t)) \neq (U_t \vee \underline{W}_t) \wedge (\underline{U}_t \vee W_t) \quad (322)$$

**2.4.2.2. The direction of causation** In general, a cause is related to its own effect in its own way and vice versa (see Mackie, 1966, p. 160) too. The effect (see Black, 1956) of this cause is itself related to its own cause in some way in which the cause is not related to its own effect (see Dummett and Flew, 1954). This can be considered as one of the reasons why the relation between cause and effect is taken to be asymmetrical.

**2.4.2.3. The priority of cause to effect** Contemporary discussions of causation are greatly influenced by the causal relation that ‘an effect  $W_t$  is causally dependent upon a cause  $U_t$ ’. However, under certain conditions (mono-causality), to say that ‘an effect  $W_t$  is causally dependent upon a cause  $U_t$ ’ is to say that ‘if a cause  $U_t$  had not occurred, then an effect  $W_t$  would not have occurred too.’ (see Lewis, David Kellogg, 1973, 1974). However, what came first, the hen or the egg, the cause or the effect?

## 2.4.3. Definition causal relationship k

**Definition 2.55 (Causal relationship k).**

Nonetheless, mathematically, the causal (Barukčić, 2011a,b, 2012) relationship (Barukčić, 1989, 1997, 2005, 2016b, 2017a,c, 2021c) between a cause  $U_t$  (German: Ursache) and an effect  $W_t$  (German: Wirkung), denoted by  $k(U_t, W_t)$ , is defined at each single (Thompson, 2006) Bernoulli trial  $t$  in terms of statistics and probability theory<sup>105</sup>,<sup>106</sup>,<sup>107</sup> as

$$k(U_t, W_t) \equiv \frac{\sigma(U_t, W_t)}{\sigma(U_t) \times \sigma(W_t)} \quad (323)$$

$$\equiv \frac{p(U_t \wedge W_t) - p(U_t) \times p(W_t)}{\sqrt{(p(U_t) \times (1 - p(U_t))) \times (p(W_t) \times (1 - p(W_t)))}}$$

where  $\sigma(U_t, W_t)$  denotes the co-variance between a cause  $U_t$  and an effect  $W_t$  at every single Bernoulli trial  $t$ ,  $\sigma(U_t)$  denotes the standard deviation of a cause  $U_t$  at the same single Bernoulli trial  $t$ ,  $\sigma(W_t)$  denotes the standard deviation of an effect  $W_t$  at same single Bernoulli trial  $t$ . Table 27 illustrates the theoretically possible relationships between a cause and an effect.

**Table 27.** Sample space and the causal relationship k

		Effect $B_t$		
		TRUE	FALSE	
Cause $A_t$	TRUE	$p(a_t)$	$p(b_t)$	$p(U_t)$
	FALSE	$p(c_t)$	$p(d_t)$	$p(\underline{U}_t)$
		$p(W_t)$	$p(\underline{W}_t)$	+1

However, even if one thinks to recognise the trace of Bravais (Bravais, 1846) (1811-1863) - Pearson's (1857-1936) "product-moment coefficient of correlation" (Galton, 1877, Pearson, 1896) inside the causal relationship k (Barukčić, 1989, 1997, 2005, 2016b, 2017a,c) both are completely different. According to Pearson: "The fundamental theorems of correlation were for the first time and almost exhaustively discussed by Bravais ('Analyse mathématique sur les probabilités des erreurs de situation d'un point.' *Memoires par divers Savans, T. IX., Paris, 1846, pp. 255-332*) nearly half a century ago." (Pearson, 1896) Neither does it make much sense to elaborate once again on the issue causation (Blalock, 1972) and correlation, since both are not identical (Sober, 2001) nor does it make sense to insist on the fact that "Pearson's philosophy discouraged him from looking too far behind phenomena." (Haldane, 1957) Whereas it is essential to consider that the causal relationship k, in contrast to Pearson's product-moment coefficient of correlation (Pearson, 1896) or to Pearson's phi

<sup>105</sup>Ilija Barukčić, "The Mathematical Formula of the Causal Relationship k," *International Journal of Applied Physics and Mathematics* vol. 6, no. 2, pp. 45-65, 2016. <https://doi.org/10.17706/ijapm.2016.6.2.45-65>

<sup>106</sup>Barukčić, Ilija. (2015). The Mathematical Formula Of The Causal Relationship k. <https://doi.org/10.5281/zenodo.3944666>

<sup>107</sup>Ilija Barukčić. The causal relationship k. MATEC Web Conf., 336 (2021) 09032 DOI: <https://doi.org/10.1051/mateconf/202133609032>

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coefficient(Pearson, 1904b), is defined at every single Bernoulli trial  $t$ . This might be a very *small* difference. However, even a small difference might determine a difference. However, in this context and in any case, this small difference *makes*(Barukčić, 2018a) the difference.

## 2.5. Axioms

Whether science needs new and obviously generally valid statements (axioms) which are able to assure the truth of theorems proved from them may remain an unanswered question. In order to be accepted, a new axiom candidate (see [Easwaran, 2008](#)) should be at least as simple as possible and logically consistent to enable advances in our knowledge of nature. The importance of axioms is particularly emphasized by Albert Einstein. “**Die wahrhaft großen Fortschritte der Naturerkenntnis sind auf einem der Induktion fast diametral entgegengesetzten Wege entstanden.**” (see [Einstein, 1919](#), p. 17). In general, *lex identitatis*, *lex contradictionis* and *lex negationis* have the potential to denote the most simple, the most general and the most far-reaching axioms of science, the foundation of our today’s and of our future scientific inquiry.

### 2.5.1. Principium identitatis (Axiom I)

**Principium identitatis** or **lex identitatis** or axiom I, is closely related to central problems of metaphysics, epistemology and of science as such. It turns out that it is more than rightful to assume that

$$+1 \equiv +1 \quad (324)$$

is true, otherwise there is every good reason to suppose that nothing can be discovered at all.

Identity as the epitome of a self-identical or of self-reference is at the same time different from difference, identity is free from difference, identity is not difference, identity is at the same time the other of itself, identity is non-identity. Identity as simple equality with itself is determined by a non-being, by a non-being of its own other, by a non-being of difference, identity is different from difference. Identity is in its very own nature different and is in its own self the opposite of itself (symmetry). It is equally

$$-1 \equiv -1 \quad (325)$$

In general, +1 and -1 are distinguished, however these distinct are related to one and the same 1. Identity as a vanishing of otherness, therefore, is this distinguishedness in one relation. It is

$$0 \equiv +1 - 1 \equiv 0 \times 1 \equiv 0 \quad (326)$$

Identity, as the unity of something and its own other is in its own self a separation from difference, and as a moment of separation might pass over into an equivalence relation which itself is reflexive, symmetric and transitive. Nonetheless, backed by thousands of years of often bitter human experience, the scientific development has taught us all that human knowledge is relative too. Even if experiments and other suitable proofs are of help to encourage us more and more in our belief of the correctness of a theory, it is difficult to prove the correctness of a theorem or of a theory et cetera once and for all. The challenge for all the science is the need to comply with Einstein’s position: “**Niemals aber kann die Wahrheit einer Theorie erwiesen werden. Denn niemals weiß man, daß auch in Zukunft eine Erfahrung bekannt werden wird, die Ihren Folgerungen widerspricht...**” ([Einstein, 1919](#)).



Albert Einstein's position translated into English: 'But the truth of a theory can never be proven. For one never knows if future experience will contradict its conclusion; and furthermore, there are always other conceptual systems imaginable which might coordinate the very same facts.' Our human experience tells us that everything in life is more or less transitory, and that nothing lasts. As a result of our knowledge and experience, several scientific theories have a glorious past to look back on, but all the glory of such scientific theories might remain in the past if scientist don't continue to innovate. In a word, theories can be refuted by time.

“No amount of experimentation can ever prove me right;  
a single experiment can prove me wrong.”

(Albert Einstein according to: [Robertson, 1998](#), p. 114)

In the light of the foregoing, it is clear that appropriate axioms and conclusions derived from the same are a main logical foundation of any 'theory'.

“**Grundgesetz (Axiome) und Folgerungen** zusammen bilden das was man **eine 'Theorie'** nennt.

”  
([Einstein, 1919](#))

However, another point is worth being considered again. One single experiment can be enough to refute a whole theory. Albert Einstein's (1879-1955) message translated into English as: *Basic law (axioms) and conclusions together form what is called a 'theory'* has still to get round. However, an axiom as a free creation of the human mind which precedes all science should be like all other axioms, as simple as possible and as self-evident as possible. Historically, the earliest documented use of **the law of identity** can be found in Plato's dialogue Theaetetus (185a) as "... each of the two is different from the other and the same as itself"<sup>108</sup>. However, Aristotle (384–322 B.C.E.), Plato's pupil and equally one of the greatest philosophers of all time, elaborated on the law of identity too. In *Metaphysica*, Aristotle wrote:

“... all things ... have some unity and identity. ”

(see [Aristotle, of Stageira \(384-322 B.C.E\), 1908](#), *Metaphysica*, Chapter IV, 999a, 25-30, p. 66)

<sup>108</sup>Plato's dialogue Theaetetus (185a), p. 104.

In *Prior Analytics*, <sup>109</sup>, <sup>110</sup> Aristotle, a tutor of Alexander, the thirteen-year-old son of Philip, the king of Macedon, is writing: “When A applies to the whole of B and of C, and is other predicated of nothing else, and B also applies to all C, A and B must be convertible. For since A is stated only of B and C, and B is predicated both of itself and of C, it is evident that B will also be stated of all subjects of which A is stated, except A itself.”<sup>111</sup>, <sup>112</sup> For the sake of completeness, it should be noted at the outset that Aristotle himself preferred **the law of contradiction** and **the law of excluded middle** as examples of fundamental axioms. Nonetheless, it is worth noting that **lex identitatis** is an axiom too, which possess the potential to serve as the most basic and equally the most simple axiom of science but has been treated by Aristotle in an inadequate manner without having any clear and determined meaning for Aristotle himself. Nonetheless, something which is really just itself is equally different from everything else. In point of fact, is such an equivalence (Degen, 1741) which everything has to itself inherent or must the same be constructed by human mind and consciousness. Can and how can something be **identical with itself** (Förster and Melamed, 2012, Hegel, Georg Wilhelm Friedrich, 1812a, Koch, 1999, Newstadt, 2015) and in the same respect different from itself. An increasingly popular view on identity is the one advocated by Gottfried Wilhelm Leibniz (1646-1716):

**“Chaque chose est ce qu’elle est. Et dans autant d’exemples qu’on voudra  
A est A,  
B est B.”**  
(Leibniz, 1765, p. 327)

or **A = A, B = B** or **+1 = +1**. In other words, a thing is what it is (Leibniz, 1765, p. 327). Leibniz’ **principium identitatis indiscernibilium** (p.i.i.), the principle of the indistinguishable, occupies a central position in Leibniz’ logic and metaphysics and was formulated by Leibniz himself in different ways in different passages (1663, 1686, 1704, 1715/16). All in all, Leibniz writes:

“C’est  
le principe des indiscernables,  
en vertu duquel  
il ne saurait exister dans la nature deux êtres identiques.  
...  
Il n’y a point deux individus indiscernables.”  
(see Leibniz, Gottfried Wilhelm, 1886, p. 45)

Exactly in complete compliance with Leibniz, Johann Gottlieb Fichte (1762 - 1814) elaborates on this subject as follows:

<sup>109</sup> Aristotle, *Prior Analytics*, Book II, Part 22, 68a

<sup>110</sup> Kenneth T. Barnes. *Aristotle on Identity and Its Problems*. Phronesis. Vol. 22, No. 1 (1977), pp. 48-62 (15 pages)

<sup>111</sup> Aristotle, *Prior Analytics*, Book II, Part 22, 68a, p. 511.

<sup>112</sup> Ivo Thomas. On a passage of Aristotle. *Notre Dame J. Formal Logic* 15(2): 347-348 (April 1974). DOI: 10.1305/ndjff/1093891315

**“Each thing is what it is ;  
it has those realities which are posited when it is posited,  
(A = A.) ”  
(Fichte, 1889)**

Georg Wilhelm Friedrich Hegel (1770 – 1831) himself objected the Law of Identity by claiming that “A = A is ... an empty tautology. ”(see [Hegel, Georg Wilhelm Friedrich, 1991](#), p. 413) provided an example of his own mechanical understanding of the Law of Identity. “the empty tautology: nothing is nothing; ... from nothing only nothing becomes ... nothing remains nothing. ”(see [Hegel, Georg Wilhelm Friedrich, 1991](#), p. 84). Nonetheless, Hegel preferred to reformulate an own version of Leibniz principium identitatis indiscernibilium in his own way by writing that “All things are different, or: there are no two things like each other. ”(see [Hegel, Georg Wilhelm Friedrich, 1991](#), p. 422). Much of the debate about identity is still a matter of controversy. This issue has attracted the attention of many authors and has been discussed by Hegel too. In this context, it is worth to consider Hegel’s radical position on identity.

“The other expression of the law of identity: A cannot at the same time be A and not-A, has a negative form; it is called the law of contradiction. ”  
([Hegel, Georg Wilhelm Friedrich, 1991](#), p. 416)

We may, usefully (see [Barukčić, 2019a](#)), state Russell’s position with respect to the identity law as mentioned in his book ‘The problems of philosophy ’ (see [Russell, 1912](#)). In particular, according to Russell,

“...principles have been singled out by tradition under the name of ‘Laws of Thought.’ They are as follows:

- (1) **The law of identity:** ‘Whatever is, is.’
- (2) **The law of contradiction:** ‘Nothing can both be and not be.’
- (3) **The law of excluded middle:** ‘Everything must either be or not be.’

These three laws are samples of self-evident logical principles, but are not really more fundamental or more self-evident than various other similar principles: for instance, the one we considered just now, which states that what follows from a true premise is true. The name ‘laws of thought’ is also misleading, for what is important is not the fact that we think in accordance with these laws, but the fact that **things behave in accordance with them;** ”

(see [Russell, 1912](#), p. 113)

Russell’s critique, that we tend too much to focus only on the formal aspects of the ‘Laws of Thoughts’ with the consequence that “... we think in accordance with these laws” (see [Russell, 1912](#), p. 113) is

justified. Judged solely in terms of this aspect, it is of course necessary to think in accordance with the ‘Laws of Thoughts’. But this is not the only aspect of the ‘Laws of Thoughts’. The other and may be much more important aspect of these ‘Laws of Thoughts’ is the fact that quantum mechanical objects or that “... things behave in accordance with them” (see [Russell, 1912](#), p. 113).

### 2.5.2. Principium contradictionis (Axiom II)

**Principium contradictionis** or **lex contradictionis** <sup>113</sup>, <sup>114</sup>, <sup>115</sup> or axiom II, the other of *lex identitatis*, the negative of *lex identitatis*, the opposite of *lex identitatis*, a complementary of *lex identitatis*, can be expressed mathematically as

$$+ 0 \equiv 0 \times 1 \equiv +1 \quad (327)$$

In addition to the above, from the point of view of mathematics, axiom II (equation 327) is equally the most simple mathematical expression and formulation of a contradiction. However, there is too much practical and theoretical evidence that a lot of ‘secured’ mathematical knowledge and rules differ too generously from real world processes, and the question may be asked whether mathematical truths can be treated as absolute truths at all. Many of the basic principle of today’s mathematics allow every single author defining the real world events and processes et cetera in a way as everyone likes it for himself. Consequentially, a resulting dogmatic epistemological subjectivism and at the end agnosticism too, after all, is one of the reasons why we should rightly heed the following words of wisdom of Albert Einstein.

**“I don’t  
believe in  
mathematics.”**

(Albert Einstein cited according to [Brian, 1996](#), p. 76)

In the long term, however, the above attitude of mathematics is not sustainable. History has taught us time and time again that objective reality has the potential to correct wrong human thinking slowly but surely, and many more than this. Objective reality has demonstrably corrected wrong human thinking again and again in the past.

<sup>113</sup>Horn, Laurence R., “Contradiction”, *The Stanford Encyclopedia of Philosophy* (Winter 2018 Edition), Edward N. Zalta (ed.), URL = <https://plato.stanford.edu/archives/win2018/entries/contradiction/>.

<sup>114</sup>Barukčić I. Aristotle’s law of contradiction and Einstein’s special theory of relativity. *Journal of Drug Delivery and Therapeutics* (JDDT). 15Mar.2019;9(2):125-43. <https://jddtonline.info/index.php/jddt/article/view/2389>

<sup>115</sup>Barukčić, Ilija. (2020, December 28). The contradiction is existing objectively and real (Version 1). Zenodo. <https://doi.org/10.5281/zenodo.4396106>

Despite all the adversities, it is necessary and crucial to consider that a self-identical as the opposite of itself is no longer only self-identity but a difference of itself from itself within itself. In other words, “All things are different, or: there are no two things like each other ... is, in fact, opposed to the law of identity ...”(see [Hegel, Georg Wilhelm Friedrich, 1991](#), p. 422) Each on its own and without any respect to the other is distinctive within itself and from itself and not only from another. As the opposite of its own something, is no longer only self-identity, but also a negation of itself out of itself and therefore a difference of itself from itself within itself. In other words, in opposition, a self-identical is able to return into simple unity with itself, with the consequence that even as a self-identical the same self-identical is inherently self-contradictory. A question of fundamental theoretical importance is, however, why should something be itself and at the same time the other of itself, the opposite of itself, not itself? Is something like this even possible at all and if so, why and how? These and similar questions have occupied many thinkers, including Hegel.

“Something is therefore  
alive only in so far as it contains contradiction within it,  
and moreover is this power to  
hold and endure the contradiction within it. ”

(see [Hegel, Georg Wilhelm Friedrich, 1991](#), p. 440)

However, as directed against identity, contradiction itself is also at the same time a source of self-changes of a self-identical out of itself.

“... contradiction  
is the root of all movement and vitality;  
it is only in so far as something has a contradiction within it  
that it moves, has an urge and activity. ”

(see [Hegel, Georg Wilhelm Friedrich, 1991](#), p. 439)

The further advance of science will throw any contribution to scientific progress of each of us back into scientific insignificance, as long as principium contradictionis is not given enough and the right attention. **The contradiction <sup>116</sup> is existing objectively and real and is the heartbeat of every self-identical.** We have reason to be delighted by the fact that very different aspects of principium contradictionis have been examined since centuries from different angles by various authors. According to Aristotle, principium contradictionis applies to everything that is, it is the first and the firmest of all principles of philosophy.

<sup>116</sup>Barukčić, Ilija. (2020, December 28). The contradiction is existing objectively and real (Version 1). Zenodo. <https://doi.org/10.5281/zenodo.4396106>

“... the same ... cannot at the same time belong and not belong to the same  
... in the same respect ... This, then, is  
the most certain of all principles ”

(see [Aristotle, of Stageira \(384-322 B.C.E\), 1908, Metaph., IV, 3, 1005b, 16–22](#))

Principium contradictionis or axiom II has many facets. As long as we follow Leibniz in this regard, we should consider that “**Le principe de contradiction est en general ...**” (Leibniz, 1765, p. 327). Scientist inevitably have false beliefs and make mistakes. In order to prevent scientific results from falling into logical inconsistency or logical absurdity, it is necessary to possess among other the methodological possibility to start a reasoning with a (logical) contradiction too. However and in contrast to the way of reasoning with inconsistent premises as proposed by para-consistent (Carnielli and Marcos, 2001, da Costa, 1974, 1958, Priest, 1998, Priest et al., 1989, Quesada, 1977) and other logic, in the absence of technical and other errors of reasoning, the contradiction itself need to be preserved. In other words, **from a contradiction does not anything follows but the contradiction itself** while the theoretical question is indeed justified “What is so Bad about Contradictions?” (Priest, 1998). Historically, **the principle of (deductive) explosion** (Carnielli and Marcos, 2001, Priest, 1998, Priest et al., 1989), coined by 12th-century French philosopher William of Soissons, demand us to accept that anything, including its own negation, can be proven or can be inferred from a contradiction. In short, according to **ex falso sequitur quodlibet**, a (logical) contradiction implies anything. Respecting the principle of explosion, the existence of a contradiction (or the existence of logical inconsistency) in a scientific theorem, rule et cetera is disastrous. However, the historical development of science shows that scientist inevitably revise the theories, false positions and claims are identified once and again, and we all make different kind of mistakes. In order to avert disproportionately great damage to science and to prevent reducing science into pure subjective belief, a negation of the principle of explosion is required. Nonetheless, a justified negation of the **ex contradictione quodlibet principle** (Carnielli and Marcos, 2001) does not imply the correctness of para consistent logic (Carnielli and Marcos, 2001, da Costa, 1974, 1958, Priest, 1998, Priest et al., 1989, Quesada, 1977) as such as advocated especially by the Peruvian philosopher Francisco Miró Quesada (Quesada, 1977) and other (Carnielli and Marcos, 2001, da Costa, 1974, 1958, Priest, 1998, Priest et al., 1989). In general, scientific theories appear to progress from lower and simpler to higher and more complex levels. However, high level theories cannot be taken for granted because high level theories are grounded on a lot of assumptions, definitions and other procedures and may rest upon too much erroneous stuff even if still not identified. Therefore, it should be considered to check at lower at simpler levels like with like.

### 2.5.2.1. Zero power zero

**Theorem 2.12.** *In general, it is*

$$+0^{+2} \equiv +0 \quad (328)$$

*is false.*

*Proof by direct proof.* The premise

$$+0 \equiv +1 \quad (329)$$

is false. In the following, any rearrangement of the premise which is free of (technical) errors, need to end up at a contradiction. In other words, the contradiction will be preserved. We obtain

$$+0 \times +0 \equiv +1 \times +0 \quad (330)$$

Equation 330 becomes

$$+0^{+2} \equiv +0 \quad (331)$$

□

### 2.5.2.2. Zero divided by zero

**Theorem 2.13.** *In general,*

$$\frac{1}{0} \equiv \frac{0}{0} \quad (332)$$

*is false.*

*Proof by direct proof.* If the premise

$$+1 \equiv +0 \quad (333)$$

is false, then the relationship

$$\frac{1}{0} \equiv \frac{0}{0} \quad (334)$$

is also false.

□

### 2.5.3. Principium negationis (Axiom III)

**Lex negationis** or axiom III, is often mismatched with simple opposition. However, from the point of view of philosophy and other sciences, identity, contradiction, negation and similar notions are equally mathematical descriptions of the most simple laws of objective reality. What sort of natural process is negation at the end? Mathematically, we define principium negationis or lex negationis or axiom III as

$$\text{Negation}(0) \times 0 \equiv \neg(0) \times 0 \equiv +1 \quad (335)$$

where  $\neg$  denotes (logical (Boole, 1854) or natural) negation (Ayer, 1952, Förster and Melamed, 2012, Hedwig, 1980, Heinemann, Fritz H., 1943, Horn, 1989, Koch, 1999, Kunen, 1987, Newstadt, 2015, Royce, 1917, Speranza and Horn, 2010, Wedin, 1990b). In this context, there is some evidence that

$$\text{Negation}(1) \times 1 \equiv \neg(1) \times 1 = 0 \quad (336)$$

Logically, it follows that

$$\text{Negation}(1) \equiv 0 \quad (337)$$

In the following we assume that axiom I is universal. Under this assumption, the following theorem follows inevitably.

**Theorem 2.14** (Zero divided by zero). *According to classical logic, it is*

$$\frac{0}{0} \equiv 1 \quad (338)$$

*Proof by direct proof.* The premise

$$1 \equiv 1 \quad (339)$$

is true. It follows that

$$\begin{aligned} 0 &\equiv 0 \\ &\equiv 0 \times 1 \end{aligned} \quad (340)$$

In the following, we rearrange the premise (see equation 335, p. 108). We obtain

$$0 \times (\text{Negation}(0) \times 0) \equiv 0 \quad (341)$$

Equation 341 changes slightly (see equation 336, p. 108). It is

$$(\text{Negation}(1) \times 1) \times (\text{Negation}(0) \times 0) \equiv 0 \quad (342)$$

Equation 342 demands that

$$(\text{Negation}(1) ) \times (\text{Negation}(0) ) \times 0 \equiv 0 \quad (343)$$



Equation 343 is logically possible (see equation 326, p. 100) only if

$$(\text{Negation}(1)) \times (\text{Negation}(0)) \equiv 1 \quad (344)$$

(see theorem 2.12, equation 328) whatever the meaning of Negation(1) or of Negation(0) might be, equation 344 demands that

$$\text{Negation}(0) \equiv \frac{1}{\text{Negation}(1)} \quad (345)$$

and that

$$\text{Negation}(1) \equiv \frac{1}{\text{Negation}(0)} \quad (346)$$

Equation 345 simplifies as (see equation 337, p. 108)

$$\begin{aligned} \text{Negation}(0) &\equiv \frac{+1}{\text{Negation}(1)} \\ &\equiv \frac{+1}{+0} \end{aligned} \quad (347)$$

It follows that

$$\neg(0) \times 0 \equiv \frac{1}{0} \times 0 \equiv \frac{0}{0} \equiv 1 \quad (348)$$

To bring it to the point. Classical logic, assumed as generally valid, demands that

$$\frac{0}{0} \equiv 1 \quad (349)$$

□

Concepts like identity, difference, negation, opposition et cetera engaged the attention of scholars at least over the last twenty-three centuries (see also [Horn, 1989](#), [Speranza and Horn, 2010](#)). As long as we first and foremost follow Josiah Royce, negatio or negation “is one of the simplest and most fundamental relations known to the human mind. For the study of logic, no more important and fruitful relation is known.” (see also [Royce, 1917](#), p. 265) But, do we really know what, for sure, what negation is? Based on what we know about negation, Aristotle (see also [Wedin, 1990a](#)) has been one of the first to present a theory of negation, which can be found in discontinuous chunks in his works the *Metaphysics*, the *Categories*, *De Interpretatione*, and the *Prior Analytics* (see also [Horn, 1989](#), p. 1). Negation (see also [Newstadt, 2015](#)) as a fundamental philosophical concept found its own very special melting point especially in Hegel’s dialectic and is more than just a formal logical process or operation which converts true to false or false to true. Negation as such is a natural process too and equally ‘**an engine of changes of objective reality**’ (see also [Barukčić, 2019a](#)). However, it remains an open question to establish a generally accepted link between this fundamental philosophical concept and an adequate counterpart in physics, mathematics and mathematical statistics et cetera. Especially the relationship between creation and conservation or *creatio ex nihilo* (see

also [Donnelly, 1970](#), [Ehrhardt, 1950](#), [Ford, 1983](#)), determination and negation (see also [Ayer, 1952](#), [Hedwig, 1980](#), [Heinemann, Fritz H., 1943](#), [Kunen, 1987](#)) has been discussed in science since ancient (see also [Horn, 1989](#), [Speranza and Horn, 2010](#)) times too. Why and how does an event occur or why and how is an event created (creation), why and how does an event maintain its own existence over time (conservation)? The development of the notion of negation leads from Aristotle to Meister Eckhart (see also [Eckhart, 1986](#)) von Hochheim (1260-1328), commonly known as Meister Eckhart (see also [Tsopurashvili, 2012](#)) or Ekehart, to Spinoza (1632 – 1677), to Immanuel Kant (1724-1804) and finally to Georg Wilhelm Friedrich Hegel (1770-1831) and other authors too. One point is worth being noted, even if it does not come as a surprise, it was especially Benedict de Spinoza (1632 – 1677) as one of the philosophical founding fathers of the Age of Enlightenment who addressed the relationship between determination and negation in his lost letter of June 2, 1674 to his friend Jarig Jelles (see also [Förster and Melamed, 2012](#)) by the discovery of his fundamental insight that “**determinatio negatio est**” (see also [Spinoza, 1674](#), p. 634). Hegel went even so far as to extended the slogan raised by Spinoza into to “Omnis determinatio est negatio” (see also [Hegel, Georg Wilhelm Friedrich, 1812b, 2010](#), p. 87). Finally, it did not take too long, and the notion of negation entered the world of mathematics and mathematical logic at least with Boole’s (see also [Boole, 1854](#)) publication in the year 1854. “Let us, for simplicity of conception, give to the symbol  $x$  the particular interpretation of men, then  $1 - x$  will represent the class of ‘not-men.’” (see also [Boole, 1854](#), p. 49). Finally, the philosophical notion negation found its own way into physics by the contributions of authors like Woldemar Voigt (see [Voigt, 1887](#)), George Francis FitzGerald (see [FitzGerald, 1889](#)), Hendrik Antoon Lorentz (see [Lorentz, 1892, 1899](#)), Joseph Larmor (see [Larmor, 1897](#)), Jules Henri Poincaré (see [Poincaré, 1905](#)) and Albert Einstein (see [Einstein, 1905](#)) by contributions to the physical notion “Lorentz factor”.

### 3. Results

#### 3.1. Without Epstein-Barr virus infection, no non-Hodkin lymphoma

Teras et al. <sup>117</sup> investigated the relationship between being EBV positive and non-Hodgkin lymphoma in general . The data and the statistical analysis are illustrated by table 28.

**Table 28.** EBV IgG Pos. and NHL (Study Teras et al. (very unfair) , 2015 ).

		NHL		
		YES	NO	
EBV IgG Pos.	YES	212	416	628
	NO	13	33	46
		225	449	674
<b>STATISTICAL ANALYSIS.</b>				
Causal relationship k =		0,0293952617		
p Value right tailed (HGD) =		0,2774570681		
p (SINE) =		0,9807121662		
$\tilde{\chi}^2$ (SINE — B <sub>t</sub> ) =		0,7511		
$\tilde{\chi}^2$ (SINE — A <sub>t</sub> ) =		3,6739		
p Value right tailed (HGD) =		0,2775		
p Value (SINE) =		0,0191030137		
<b>RELATIVE RISK (RR).</b>				
RR (nc) =		1,1945		
RR (sc) =		1,0170		
<b>ADDITIONAL MEASURES.</b>				
OR =		0,3635		
IOR =		0,0112		
<b>STUDY DESIGN.</b>				
p(IOU)=		0,265578635		
p(IOI)=		0,597922849		

<sup>117</sup>Teras LR, Rollison DE, Pawlita M, Michel A, Brozy J, de Sanjose S, Blase JL, Gapstur SM. Epstein-Barr virus and risk of non-Hodgkin lymphoma in the cancer prevention study-II and a meta-analysis of serologic studies. *Int J Cancer*. 2015 Jan 1;136(1):108-16. doi: 10.1002/ijc.28971. Epub 2014 Jun 5. PMID: 24831943. see also: <https://pubmed.ncbi.nlm.nih.gov/24831943/>

### 3.2. Without Epstein-Barr virus infection, no non-Hodkin lymphoma (fair)

Teras et al. <sup>118</sup> investigated the relationship between being EBV positive and NHL . The data and the statistical analysis under conditions of a fair study design are illustrated by table 29.

**Table 29.** EBV positive and NHL (Study Teras et al. (fair) , 2015 ).

		NHL		
		YES	NO	
EBV positive	YES	212	1362	1574
	NO	13	212	225
		225	1574	1799

#### STATISTICAL ANALYSIS.

Causal relationship k =	0,0769109135
p Value right tailed (HGD) =	0,0003360340
<b>p (SINE) =</b>	<b>0,9927737632</b>
$\tilde{\chi}^2$ (SINE — B <sub>t</sub> ) =	0,7511
$\tilde{\chi}^2$ (SINE — A <sub>t</sub> ) =	0,7511
p Value right tailed (HGD) =	0,0003
p Value (SINE) =	0,0072001903

#### RELATIVE RISK (RR).

RR (nc) =	2,3312
RR (sc) =	1,0889

#### ADDITIONAL MEASURES.

OR =	0,2357
IOR =	0,0769

#### STUDY DESIGN.

p(IOU)=	0
p(IOI)=	0,749861034

<sup>118</sup>Teras LR, Rollison DE, Pawlita M, Michel A, Brozy J, de Sanjose S, Blase JL, Gapstur SM. Epstein-Barr virus and risk of non-Hodgkin lymphoma in the cancer prevention study-II and a meta-analysis of serologic studies. *Int J Cancer*. 2015 Jan 1;136(1):108-16. doi: 10.1002/ijc.28971. Epub 2014 Jun 5. PMID: 24831943. see also: <https://pubmed.ncbi.nlm.nih.gov/24831943/>

### 3.3. Without Epstein-Barr virus infection, no DLBCL

DLBCL: diffuse large B-cell lymphoma; Teras et al. <sup>119</sup> investigated the relationship between being EBV positive and diffuse large B-cell lymphoma . The data and the statistical analysis are illustrated by table 30.

**Table 30.** EBV positive and DLBCL (Study Teras et al. (unfair) , 2015 ).

		DLBCL		
		YES	NO	
EBV positive	YES	65	416	481
	NO	2	33	35
		67	449	516

#### STATISTICAL ANALYSIS.

Causal relationship k = 0,0583441872  
 p Value right tailed (HGD) = 0,1404865520  
**p (SINE) = 0,9961240310**  
 $\tilde{\chi}^2$  (SINE — B<sub>t</sub>) = 0,0597  
 $\tilde{\chi}^2$  (SINE — A<sub>t</sub>) = 0,1143  
 p Value right tailed (HGD) = 0,1405  
 p Value (SINE) = 0,0038684671

#### RELATIVE RISK (RR).

RR (nc) = 2,3649  
 RR (sc) = 1,0471

#### ADDITIONAL MEASURES.

OR = 0,1899  
 IOR = 0,0407

#### STUDY DESIGN.

p(IOU)= 0,062015504  
 p(IOI)= 0,802325581

<sup>119</sup>Teras LR, Rollison DE, Pawlita M, Michel A, Brozy J, de Sanjose S, Blase JL, Gapstur SM. Epstein-Barr virus and risk of non-Hodgkin lymphoma in the cancer prevention study-II and a meta-analysis of serologic studies. *Int J Cancer*. 2015 Jan 1;136(1):108-16. doi: 10.1002/ijc.28971. Epub 2014 Jun 5. PMID: 24831943. see also: <https://pubmed.ncbi.nlm.nih.gov/24831943/>

### 3.4. Without Epstein-Barr virus infection, no FL

Teras et al. <sup>120</sup> investigated the relationship between being EBV positive and follicular lymphoma . The data and the statistical analysis are illustrated by table 31.

**Table 31.** EBV positive and FL (Study Teras et al. (unfair) , 2015 ).

		FL		
		YES	NO	
EBV positive	YES	42	416	458
	NO	2	33	35
		44	449	493

#### STATISTICAL ANALYSIS.

Causal relationship k =	0,0311310303
p Value right tailed (HGD) =	0,3761798637
<b>p (SINE) =</b>	0,9959432049
$\tilde{\chi}^2$ (SINE — B <sub>t</sub> ) =	0,0909
$\tilde{\chi}^2$ (SINE — A <sub>t</sub> ) =	0,1143
p Value right tailed (HGD) =	0,3762
p Value (SINE) =	0,0040485775

#### RELATIVE RISK (RR).

RR (nc) =	1,6048
RR (sc) =	1,0303

#### ADDITIONAL MEASURES.

OR =	0,1521
IOR =	0,0275

#### STUDY DESIGN.

p(IOU)=	0,018255578
p(IOI)=	0,839756592

<sup>120</sup>Teras LR, Rollison DE, Pawlita M, Michel A, Brozy J, de Sanjose S, Blase JL, Gapstur SM. Epstein-Barr virus and risk of non-Hodgkin lymphoma in the cancer prevention study-II and a meta-analysis of serologic studies. *Int J Cancer*. 2015 Jan 1;136(1):108-16. doi: 10.1002/ijc.28971. Epub 2014 Jun 5. PMID: 24831943. see also: <https://pubmed.ncbi.nlm.nih.gov/24831943/>

### 3.5. Without Epstein-Barr virus infection, no CLL/SLL

Teras et al. <sup>121</sup> investigated the relationship between being EBV positive and chronic lymphocytic leukemia/small lymphocytic lymphoma . The data and the statistical analysis are illustrated by table 32.

**Table 32.** EBV positive and CLL/SLL (Study Teras et al. (unfair) , 2015 ).

		CLL/SLL		
		YES	NO	
EBV positive	YES	61	416	477
	NO	5	33	38
		66	449	515

#### STATISTICAL ANALYSIS.

Causal relationship k =	-0,0028908708
p Value right tailed (HGD) =	0,6426320619
<b>p (SINE) =</b>	0,9902912621
$\tilde{\chi}^2$ (SINE — B <sub>t</sub> ) =	0,3788
$\tilde{\chi}^2$ (SINE — A <sub>t</sub> ) =	0,6579
p Value right tailed (HGD) =	0,6426
p Value (SINE) =	0,0096617602

#### RELATIVE RISK (RR).

RR (nc) =	0,9719
RR (sc) =	0,9976

#### ADDITIONAL MEASURES.

OR =	0,1825
IOR =	-0,0021

#### STUDY DESIGN.

p(IOU)=	0,054368932
p(IOI)=	0,798058252

<sup>121</sup>Teras LR, Rollison DE, Pawlita M, Michel A, Brozy J, de Sanjose S, Blase JL, Gapstur SM. Epstein-Barr virus and risk of non-Hodgkin lymphoma in the cancer prevention study-II and a meta-analysis of serologic studies. *Int J Cancer*. 2015 Jan 1;136(1):108-16. doi: 10.1002/ijc.28971. Epub 2014 Jun 5. PMID: 24831943. see also: <https://pubmed.ncbi.nlm.nih.gov/24831943/>

### 3.6. Without Epstein-Barr virus infection, no other NHL

Teras et al. <sup>122</sup> investigated the relationship between being EBV positive and other NHL . The data and the statistical analysis are illustrated by table 33.

**Table 33.** EBV positive and Other NHL (Study Teras et al. (unfair) , 2015 ).

		Other NHL		
		YES	NO	
EBV positive	YES	44	416	460
	NO	2	33	35
		46	449	495

#### STATISTICAL ANALYSIS.

Causal relationship k =	0,0339998237
p Value right tailed (HGD) =	0,3474328248
<b>p (SINE) =</b>	0,9959595960
$\tilde{\chi}^2$ (SINE — B <sub>t</sub> ) =	0,0870
$\tilde{\chi}^2$ (SINE — A <sub>t</sub> ) =	0,1143
p Value right tailed (HGD) =	0,3474
p Value (SINE) =	0,0040322526

#### RELATIVE RISK (RR).

RR (nc) =	1,6739
RR (sc) =	1,0324

#### ADDITIONAL MEASURES.

OR =	0,1556
IOR =	0,0293

#### STUDY DESIGN.

p(IOU)=	0,022222222
p(IOI)=	0,836363636

<sup>122</sup>Teras LR, Rollison DE, Pawlita M, Michel A, Brozy J, de Sanjose S, Blase JL, Gapstur SM. Epstein-Barr virus and risk of non-Hodgkin lymphoma in the cancer prevention study-II and a meta-analysis of serologic studies. *Int J Cancer*. 2015 Jan 1;136(1):108-16. doi: 10.1002/ijc.28971. Epub 2014 Jun 5. PMID: 24831943. see also: <https://pubmed.ncbi.nlm.nih.gov/24831943/>



### 3.7. Without Epstein-Barr virus infection, no other NHL

Kimberly A Bertrand et al. <sup>123</sup> investigated the relationship between EBV and NHL. The data and the statistical analysis are illustrated by table 34.

**Table 34.** EBV positive and NHL (Study Bertrand et al. (vera unfair), 2010 ).

		NHL		
		YES	NO	
EBV positive	YES	319	629	948
	NO	21	33	54
		340	662	1002
<b>STATISTICAL ANALYSIS.</b>				
Causal relationship k =		-0,0249855706		
p Value right tailed (HGD) =		0,8265017037		
p (SINE) =		0,9790419162		
$\tilde{\chi}^2$ (SINE — B <sub>t</sub> ) =		1,2971		
$\tilde{\chi}^2$ (SINE — A <sub>t</sub> ) =		8,1667		
p Value right tailed (HGD) =		0,8265		
p Value (SINE) =		0,0207399895		
<b>RELATIVE RISK (RR).</b>				
RR (nc) =		0,8653		
RR (sc) =		0,9875		
<b>ADDITIONAL MEASURES.</b>				
OR =		0,3513		
IOR =		-0,0083		
<b>STUDY DESIGN.</b>				
p(IOU)=		0,285429142		
p(IOI)=		0,606786427		

The data of Bertrand et al. are self-contradictory ( $k < 0$ , conditio sine qua non relationship significant) and have not been considered for our conclusions.

<sup>123</sup>Bertrand KA, Birmann BM, Chang ET, Spiegelman D, Aster JC, Zhang SM, Laden F. A prospective study of Epstein-Barr virus antibodies and risk of non-Hodgkin lymphoma. *Blood*. 2010 Nov 4;116(18):3547-53. doi: 10.1182/blood-2010-05-282715. Epub 2010 Jul 20. PMID: 20647565; PMCID: PMC2981477.

### 3.8. Without Epstein-Barr virus infection, no other NHL (fair study design)

Under conditions of a fair study design (  $a = d$  ), even the study of Kimberly A Bertrand et al. <sup>124</sup> provides evidence of the relationship between EBV and NHL. The data and the statistical analysis are illustrated by table 35.

**Table 35.** EBV positive and NHL (Study Bertrand et al. (very unfair) , 2010 ).

		NHL		
		YES	NO	
EBV positive	YES	319	3084	3403
	NO	21	319	340
		340	3403	3743

#### STATISTICAL ANALYSIS.

Causal relationship  $k = 0,0319761110$   
 p Value right tailed (HGD) = 0,0273116310  
**p (SINE) = 0,9943895271**  
 $\tilde{\chi}^2$  (SINE —  $B_t$ ) = 1,2971  
 $\tilde{\chi}^2$  (SINE —  $A_t$ ) = 1,2971  
 p Value (SINE) = 0,0055947636

#### RELATIVE RISK (RR).

RR (nc) = 1,5177  
 RR (sc) = 1,0353

#### ADDITIONAL MEASURES.

OR = 0,1705  
 IOR = 0,0320

#### STUDY DESIGN.

p(IOU)= 0  
 p(IOI)= 0,818327545

<sup>124</sup>Bertrand KA, Birmann BM, Chang ET, Spiegelman D, Aster JC, Zhang SM, Laden F. A prospective study of Epstein-Barr virus antibodies and risk of non-Hodgkin lymphoma. *Blood*. 2010 Nov 4;116(18):3547-53. doi: 10.1182/blood-2010-05-282715. Epub 2010 Jul 20. PMID: 20647565; PMCID: PMC2981477.

#### 4. Discussion

Most of the studies that came into question for being re-analysed presented self-contradictory data or the study design was inadequate. For these reasons, we specifically reviewed the study of Terras et al. and used the data of this study as the basis of our conclusion. Nonetheless, are there really good reasons for believing that one single study is enough to find out the truth? Unfortunately, such a question cannot be answered in a general way, neither to the positive nor to the negative. Various unfavourable factors influencing the quality of a single study cannot be excluded completely. At the very least, for this reason, it is very difficult to rely exclusively on a single study. Indeed, it appears extremely appropriate to consider independent investigations by several study groups in order to underline our confidence into a certain relationship. In spite of everything that has been said, theoretically, one single study is enough to detect or to establish an everlasting relationship. A study which has been conducted under normal conditions, free of ideological influence and other negative factors, done to the best of knowledge of the authors, has the potential to help us to recognise how this world is working. To put it concisely, even one single study can be enough to find out the truth. As an example, let a study group investigate the relationship between a burning wax candle and gaseous oxygen. At the end and regardless of the sample size, such a study group should be able to find out that without gaseous oxygen, no burning wax candle. Other study groups should be able to confirm this relationship. We must emphasise, however, that there has not been that much wrong with the data analysed in this article. However and despite the strengths of the results of this study noted above, at least one weakness should be taken into consideration when interpreting the results. A key complication of the analysis are the missing data about sensitivity and specificity of the method used to identify an EBV infection. We have no alternative but to demand that other shortcomings should not be overlooked too. There were 13 individuals out of 674 persons who were EBV negative but still suffered from an NHL. All of this theoretically rules out the possibility that EBV is a necessary condition of the NHL. These cases would have to be examined more closely, which unfortunately is not so easy to do within the scope of this investigation. Nevertheless, the question worth being asked is, why was this so? Was it due to objective factors? Was the sensitivity and specificity of the method used to determine individuals as EBV positive insufficient or was it due to subjective factors? Have inexperienced doctors diagnosed NHL when in fact there was none et cetera? Future research into NHL should be very specific in this regard. All things considered, we have to stick with what we have. We rightly have a statistical tool, the **significance level**  $\alpha$  which is necessary especially because of such adversities too. Lastly (see table 28), it should be pointed out that all that remains is to come to the following and inescapable conclusion.

#### 5. Conclusion

**Without** an Epstein-Barr virus infection, **no** non-Hodkin lymphoma (P Value = 0,0191030137). Within EBV, the cause of NHL will be found.

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## Acknowledgments

No funding or any financial support by a third party was received. Thanks to Overleaf, Zotero, Gnuplot, Zenodo, TeX Shop and other.

## 6. Patient consent for publication

Not required.

## Conflict of interest statement

No conflict of interest to declare.

## Private note

The definition section of a paper need not and does not necessarily contain new scientific aspects. Above all, it also serves to better understand a scientific publication, to follow every step of the arguments of an author and to explain in greater details the fundamentals on which a publication is based. Therefore, there is no objective need to force authors to reinvent a scientific wheel once and again unless such a need appears obviously factually necessary. The effort to write about a certain subject in an original way in multiple publications does not exclude the necessity simply to cut and paste from an earlier work, and has nothing to do with self-plagiarism. However, such an attitude cannot simply be transferred to the sections' introduction, results, discussion and conclusions et cetera.

Erratum: There is no good reason to believe that glyphosate is a cause, or even the cause, of the NHL. <sup>125</sup>

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<sup>125</sup>Barukčić I. Glyphosate and Non-Hodgkin lymphoma: No causal relationship. JDDT [Internet]. 15Feb.2020 [cited 3Jul.2022];10(1-s):6-9. Available from: <https://jddtonline.info/index.php/jddt/article/view/3856>

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I was born October, 1<sup>st</sup> 1961 in Novo Selo, Bosnia and Herzegovina, former Yugoslavia. I am of Croatian origin. From 1982-1989 C.E., I studied human medicine at the University of Hamburg, Germany. Meanwhile, I am working as a specialist of internal medicine. My basic field of research since my high school days at the Wirtschaftsgymnasium Bruchsal, Baden Württemberg, Germany is the mathematization of the relationship between a cause and an effect valid without any restriction under any circumstances including the conditions of classical logic, probability theory, quantum mechanics, special and general theory of relativity, human medicine et cetera. I endeavour to investigate positions of quantum mechanics, relativity theory, mathematics et cetera, only insofar as these positions put into question or endanger **the general validity of the principle of causality**.



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